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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Next Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

October 31, November 1, 2, 1957

Miami, Florida

EDWARD B. SPALDING

Birmingham, Michigan

We are pleased to dedicate this issue of the Journal of Periodontology to one of the stalwarts of the dental profession, Dr. Edward B. Spalding of Birmingham, Michigan. Dr. Spalding, who has been practicing dentistry for sixty-four years and still goes to his office every day, started as far back as 1892 to fight for recognition for periodontology. Working constantly with his wife, Dr. Grace Rogers Spalding, who was the first editor of the Journal of Periodontology, he has been militant in his defense of the importance of the treatment of periodontal disease. The stature gained by the Academy of Periodontology and the respect given the specialty of periodontology, is due to idealists like Dr. Spalding who continue to insist that periodontology deserves the careful attention of every practicing dentist. Dr. Spalding served as president of the Academy, when the meeting was held in Atlantic City in 1937, and was elected a Fellow of the Academy in 1953. The Academy is indebted to Dr. Spalding for his leadership and sage advice.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-SIX

The Results Obtained with an Unembellished Gingivectomy Technic in a Clinical Study in Humans*

by IRVING GLICKMAN, B.S., D.M.D.,** *Boston, Mass.*

A VARIETY of preliminary or adjunctive procedures have been recommended as necessary for the attainment of most desirable results when the gingivectomy technic is used for eradicating periodontal pockets. Preliminary oral prophylaxis including the removal of all calcareous deposits and packing the pockets with loosely rolled cotton spindles or strings soaked in surgical cement are recommended before the gingivectomy is performed.[1] Applications of a drug such as silver nitrate, zinc chloride or trichloracetic acid are suggested when the periodontal pack is changed after the tissue has been resected.[2] Because physiologic gingival architecture is important to gingival health it has been suggested that the gingivectomy be enhanced by "plastic" procedures to properly fashion the gingiva.[3] Technics entailing the use of periodontal knives, electrosurgery[4] and even rotating coarse diamond stones[5] have been recommended for creating proper contour in the gingiva.

It has been our concept that the gingivectomy is a direct, unembellished method for eliminating periodontal pockets which consists of two stages, (1) resection of the diseased pocket wall and (2) removal of calculus and smoothing the tooth surface, performed in one operation and followed by the application of a periodontal pack. It is a technic which does not ordinarily require preliminary or post-gingivectomy procedures or the use of drugs. We were concerned with the possibility that presentations which focussed attention upon adjunctive procedures might inadvertently or intentionally create the impression the gingivectomy could not be depended upon to attain desirable results unless such embellishments were included. We felt that a useful purpose would be served by a presentation of the results obtainable with an unembellished gingivectomy technic. The following is a report of a clinical study in which the unembellished gingivectomy was put to the test by which all treatment must ultimately be judged, namely that of clinical performance.

Material

Two hundred and fifty patients with chronic periodontal disease of varied severity were treated by gingivectomy. In 200 of the patients the mouth was

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divided into quadrants, treated singly at weekly intervals. In 50 patients a full mouth gingivectomy was performed in one operation. All patients were treated by the same individual and were under observation for periods of from three months to seven years.

The patients consisted of males and females from the age of 18 to 63. The conditions treated included chronic marginal gingivitis, periodontal pockets with destruction of alveolar bone, bifurcation and trifurcation involvement and gingival enlargement. Severity of involvement was evaluated in terms of pocket depth and the level of attachment of the base of the pocket on the tooth surface. Pockets were classified as "deep" when the calculary deposits on the root surface could not be completely visualized by deflecting the pocket wall with a probe or a blast of warm air.

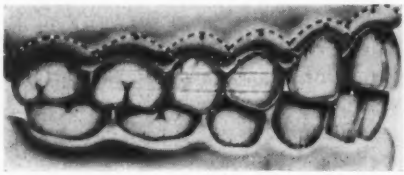

Fig. 1. Outline of gingivectomy incision just apical to the bottom of the pockets indicated by pinpoint guide markings.

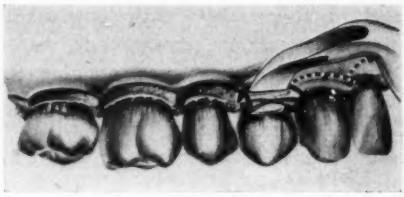

Fig. 2. Detaching the excised gingival margin along discontinuous incision using a Kirkland instrument.

Among the patients was a group of fifty individuals with only slight to moderate pocket formation, without appreciable destruction of the underlying tissues or denudation of the root surface. Ordinarily, this type of condition would not be treated with gingivectomy. The patients were included in the study to determine whether there would be disfigurement of the gingiva or denudation of tooth surface if the gingivectomy were performed under such circumstances.

Technic

The Kirkland gingivectomy technic, with some modification,[6] was employed for all patients. The operation performed under local anesthesia, consisted of the following procedures, using an aspirator to facilitate visualization of the tooth surfaces: determination of the depth and course of each pocket using a periodontal probe and making pinpoint guide marks on the facial, lingual and interproximal gingiva (Fig. 1); resection of the pocket wall at a level apical to the base of each pocket (Fig. 2); removal of the soft granulation tissue, the alveolar bone was not filed or trimmed; removal of all deposits from the teeth and smoothing of the root surfaces (Fig. 3); placing a periodontal pack,* removal of the periodontal pack after one week; patient instruction in oral physiotherapy; adjunct procedures when indicated such as removal of overhanging restoration margins and elimination of food impaction areas. Semilunar incisions, either the continuous or discontinuous type were used on both the facial and lingual surfaces (Fig. 2). The incisions were made at a bevel of approximately 45° to the tooth surface (Fig. 4) except in areas of pronounced gingival enlargement where the bevel was accentuated (Fig. 5). The outline of the incisions conformed to the contour of the base of the pockets. No effort was made to preserve a regular incision outline by permitting portions of a pocket to remain in situ.

*Powder, four parts of an equal mixture by weight of zinc oxide and powdered rosin, one part tannic acid flakes, Liquid, equal mixture of peanut oil and eugenol plus rosin.

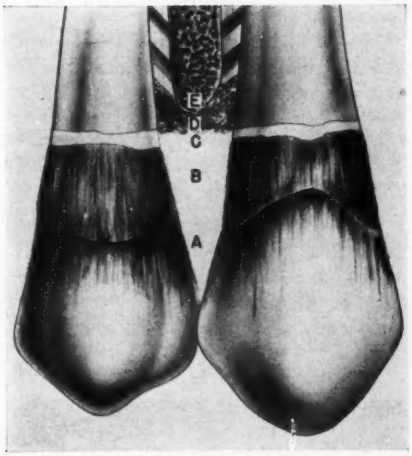

Fig. 3. Diagrammatic representation of the structures which confront the operator after the gingiva is removed. A—remnants of supragingival calculus on the crown. B—subgingival calculus on the root surface which formed the wall of the periodontal pocket. C—clear space where the epithelium was attached to the root. D—soft friable granulation tissue. E—bone.

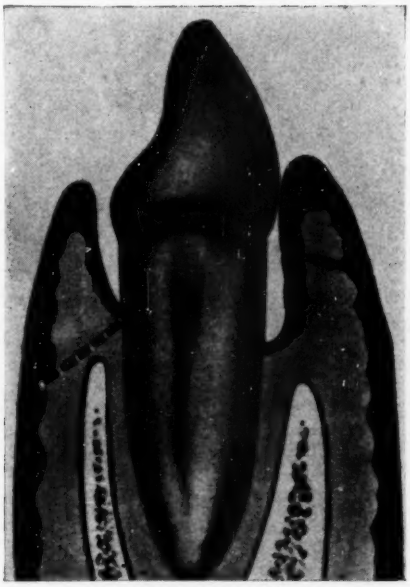

Fig. 4. Labio-lingual section through the mandibular canine. Left side, correct location of the line of incision below the bottom of the pocket at a bevel of approximately 45°. Right side, incorrect incision which fails to resect entire pocket wall.

Other than the semilunar outline and bevel of the incision, there was no shaping of the gingiva or fashioning of gingival architecture with diamond stone or electrosurgery.

Each gingivectomy, whether a quadrant or the entire mouth, was performed as a complete operation. Resection of the gingiva was followed immediately by thorough removal of the calculus and smoothing of the root without an intervening "waiting" period. Particular emphasis was placed upon removal of calculus in its entirety. No drugs were used in the gingivectomy procedure, nor was there any pre-treatment scaling or drug therapy in an attempt to reduce the depth of periodontal pockets before the gingivectomy.

Scope of the Study

The study was confined to clinical evaluation of the unembellished gingivectomy operation for the elimination of periodontal pockets. Other important aspects of overall patient management such as diagnosis, determination of prognosis and treatment planning are not included in the report in order to avoid distracting from its solitary purpose.

Observations were made of each patient at specific intervals throughout the course of treatment. The following clinical criteria were used in evaluating the effects of the gingivectomy:

1. *Dependability of the gingivectomy:* Effectiveness in restoring gingival health and attaining complete eradication of periodontal pockets;

2. *Longevity:* Duration of the persistence of physiologic sulcus depth and gingival health;

3. *Applicability of the gingivectomy technic:* The frequency and extent of complications such as patient discomfort, hemorrhage or localized infection with or without systemic complications;

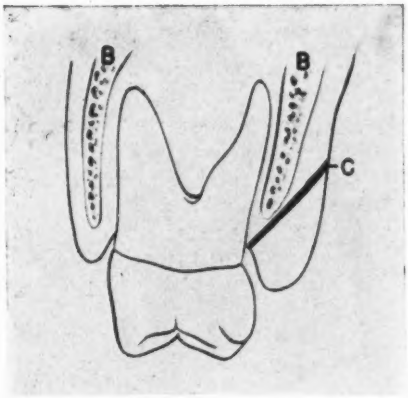

Fig. 5. Accentuated bevel (c) just above the bone (b) when gingiva along palatal surface of maxillary molar presents bulbous enlargement.

4. *Post-treatment root sensitivity:* The occurence and duration of root sensitivity to thermal changes and tactile stimulation;

5. *Post-treatment aesthetics combined with gingival health:* Evaluation of post-treatment aesthetics was a major phase of the study.

Clinical Findings

1. Dependability of the gingivectomy.

Dependability of the gingivectomy was evaluated in terms of effectiveness in restoring gingival health and attaining complete eradication of periodontal pockets. Physiologic sulcus depth up to 2 mm. and the restoration of gingival health were consistent findings. Exceptions to this rule could be traced to one or more of the following causes; incomplete removal of calculus, inadequate curetting of the root surface, incomplete elimination of other sources of local irritation such as overhanging fillings and food impaction areas, or insufficient attention to oral physiotherapy by the patient.

In all cases, the elimination of periodontal pockets required the fastidious removal of deposits on the root followed by careful smoothing of the root surface. By removing the diseased soft tissue wall of the pocket the gingivectomy provided accessibility and visibility and made complete removal of the calculus and curetting of the root surface possible.

2. Longevity: Duration of the persistence of the physiologic gingival sulcus and gingival health.

The period of time individual patients were under observation varied from three months to seven years. The most common cause of relapse was failure on the part of the patient to maintain a regime of gingival massage and interdental stimulation. In such cases, removal of accumulated debris and reestablishment of efficient oral physiotherapy were followed by a restoration of gingival health. Observations made as long as seven years after the completion of treatment attested to the health prolonging benefits from gingivectomy (Fig. 6).

Fig. 6. Above. Before treatment. Patient with chronic marginal gingivitis and moderate pocket formation. Note bulbous gingival enlargement in mandibular anterior area. Below. Seven years after gingivectomy, showing persistence of improvement in condition of the gingiva.

The thorough elimination of periodontal

pockets and the restoration of gingival health attained with the gingivectomy resulted in a gingival contour which assisted in the maintenance of a healthy periodontium. The rounded knivelike margin at the junction of the treated gingiva and the tooth surface and the firm pointed interdental papillae which resulted from healing following gingivectomy were conducive to unimpaired excursion of the food in the course of mastication (Fig. 7). This provided the benefits of natural gingival massage and at the same time minimized the accumulation of food debris which could initiate gingival inflammation.

3. Applicability of the gingivectomy technic.

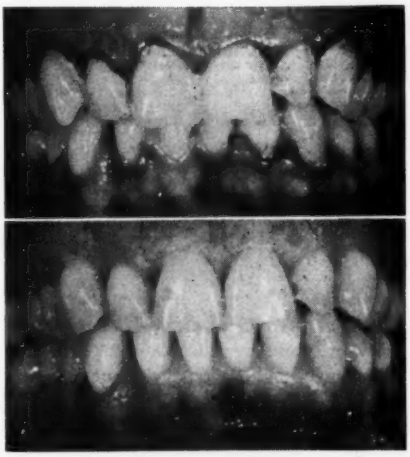

Fig. 7. Above. Before treatment. Chronic marginal gingivitis with pocket formation and distortion of the marginal and interproximal gingival contours. Below. Three months after gingivectomy. Pockets eliminated, gingival health and physiologic gingival contour restored.

Under the heading of applicability we considered the nature of the problems incidental to the use of the gingivectomy technic. As noted earlier, quadrants of the mouth were treated at weekly intervals, except in 50 cases in which full mouth gingivectomy was performed in one operation. Undisturbed healing and uneventful recovery in the first post-operative week were

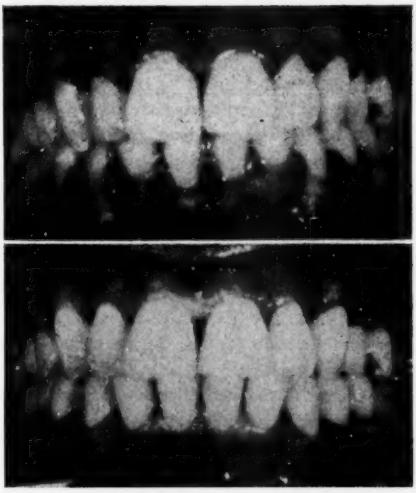

Fig. 8. Chronic marginal gingivitis in patient with past history of episodes of acute gingival disease. Note blunting of interproximal papillae in right anterior maxilla and left mandibular premolar areas. Below. Eight months after gingivectomy, gingival health and excellent contour are restored. Note well formed interproximal papillae in areas previously destroyed by disease.

the general rule. In approximately twenty-five per cent of the cases, the patients reported some discomfort "when the novocaine wore off." This was alleviated satisfactorily by 10 grains of aspirin. In rare instances was there any complaint of pain following this initial discomfort.

Bleeding in the course of the operation presented no problem. Use of the aspirator maintained a clear field and permitted visibility of the root surfaces. At the completion of the operation the periodontal pack was applied after the clot formed. In no cases was it necessary to use a hemostatic to control bleeding.

Post-operative infection rarely occurred. In isolated cases, pinpoint vesicular lesions were seen on the mucous membrane adjacent to the field of operation. These were caused by virus infection in areas where the surface continuity of the mucous membrane was disturbed by the pressure of the "finger rest" during scaling. Such areas

were sensitive to thermal and tactile stimulation, and usually healed in one week.

Loosening and breaking away of pieces of the pack frequently occurred in the latter part of the first post-operative week. This was of little consequence to the patient. Disturbance of the pack in the first or second post-operative day occurred less frequently. It was attended by some discomfort which was easily remedied by repacking the involved area. Loosening and breaking of the pack in the early post-operative period could be prevented by avoiding excess when placing the pack. It was advisable to have the patient remain in the chair for about 15 minutes after the pack was applied. Excessive pack was forced onto the occlusal surface by the pressure of the tongue and cheek muscles and was removed with a scaler. This eliminated obstructive bulk which would interfere with the occlusion and pry away the pack during chewing.

In the 50 cases in which the full mouth gingivectomy was performed in one operation, it was found more satisfactory to have the patient hospitalized for 24 hours. The operation was performed at 8 a.m. under local anesthesia and the patient was discharged at approximately 3 p.m. The patient was admitted to the hospital the previous afternoon to permit medical checkup and premedication.

The post-operative and follow-up care for these patients was the same as that following treatment of one quadrant of the mouth. The patient returned one week after the operation for removal of the pack, instruction in oral physiotherapy and correction of any environmental conditions which might impair healing or be detrimental to the maintenance of periodontal health. The full mouth gingivectomy operation presented no unique post-operative sequellae. The impairment in chewing was accepted as small inconvenience compared with the advantage of eliminating disease of long standing in one session.

4. Post-treatment root sensitivity.

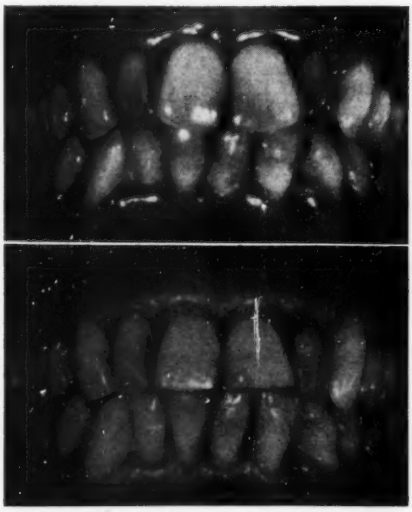

Fig. 9. Above. Before treatment. Extensive pocket formation with destruction of underlying tissue and bulbous enlargement of the inflamed gingiva. Below. Twelve months after gingivectomy. The periodontal pockets are eliminated and portions of the root surface are now visible. These were denuded by disease but obscured from view by the inflamed gingiva before treatment. Superficial calculus is present on root surfaces of mandibular anterior teeth at time patient returned for one year check-up visit.

Immediately after the pack was removed there was sensitivity to thermal and tactile stimulation. This diminished progressively so that at the end of a two-week period it was usually not significant. Root sensitivity which persisted beyond this period was alleviated by a 2% flavored aqueous solution of sodium fluoride to be used by the patient instead of a dentifrice during tooth brush massage. Satisfactory results were generally obtained by the time eight ounces of this solution had been used. In isolated cases it was necessary to supplement this regime by burnishing a paste of sodium fluoride, kaolin and glycerin (equal parts by weight) on tooth surfaces with persistent sensitivity.

5. Post-treatment esthetics combined with gingival health.

Post-treatment esthetics was evaluated in terms of the color, consistency and contour of the gingiva and the position of the gin-

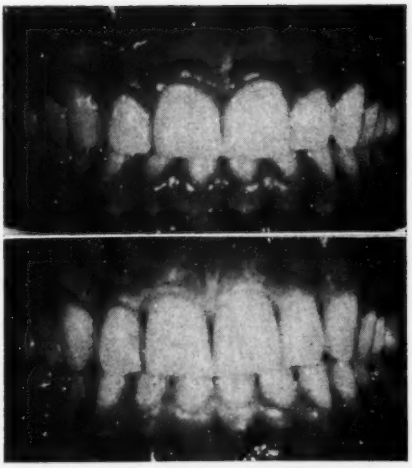

Fig. 10. Above. Before treatment. Slight marginal gingivitis in patient treated with gingivectomy for experimental purposes. Below. Eleven months after gingivectomy. There is marked improvement in the appearance of the gingiva with restoration of gingival contour conducive to the maintenance of health.

gival sulcus on the tooth, or the amount of visible root surface. In no instance was the case considered as ready for final evaluation unless all pockets were completely eliminated. Observations were made at specific phases in the overall treatment regime as follows:

(a) *Appearance before treatment*—To provide ample material for evaluation the esthetic results following gingivectomy, patients with all degrees of pocket formation and tissue destruction from periodontal disease were studied. All gradations of denudation of the root surface were represented before any treatment was undertaken. The cases varied from slight to moderate pocket formation (Fig. 8), to deep pockets with bone loss and denudation of root surface (Fig. 9). The 50 patients with comparatively early inflammatory changes, included for the purpose of determining whether undesirable esthetics would result if the gingivectomy were performed under such circumstances, presented only slight alterations in gingival appearance (Fig. 10).

(b) *Appearance after the pocket wall was resected, before the periodontal pack was applied.* The soft tissue wall of all pockets was resected below the epithelial attachment and the outline of the incision followed the pocket contours (Fig. 11, top right). Resection of the pocket wall exposed tooth surface which had been denuded by pocket formation but which had been obscured by the bulk of the diseased gingiva. The incision was marked by a clean-cut bleeding surface, without adherent tabs of tissue.

(c) *Appearance at the time of pack removal, one week after gingivectomy*—The cut surface was covered with a friable, adherent grey mesh beneath which an undertone of varied degrees of redness could be seen (Fig. 11, bottom left). The gingiva was slightly edematous and the angle formed by the cut surface and the gingival mucosa was rounded. In varied degrees, portions of tooth surface which was visible at the time of incision, were now partially covered.

(d) *Appearance at the termination of the treatment regime*—The interval between this observation and the time of operation varied from 8 to 12 weeks. No final observations were made until all signs of gingival disease or pocket formation had disappeared. At the time of final observation the gingiva was healthy, pale pink and firm with rounded, knivelike adherent margins. Blunted, partially destroyed interproximal gingival papillae were replaced by well formed, firm tissue (Figs. 8 & 11). Healthy gingiva invariably was attached to the tooth at a level in some degree coronal to the line of incision (Fig. 11, top right and bottom right).

The gingivectomy resulted in improved gingival esthetics. The amount of root surface visible after healing depended upon the extent of tissue destruction before treatment was instituted (Compare Figs. 8, 9, and 10). In the patients with gingival change but no appreciable destruction of the underlying tissues, no root surface was visible after treatment, indicating that the

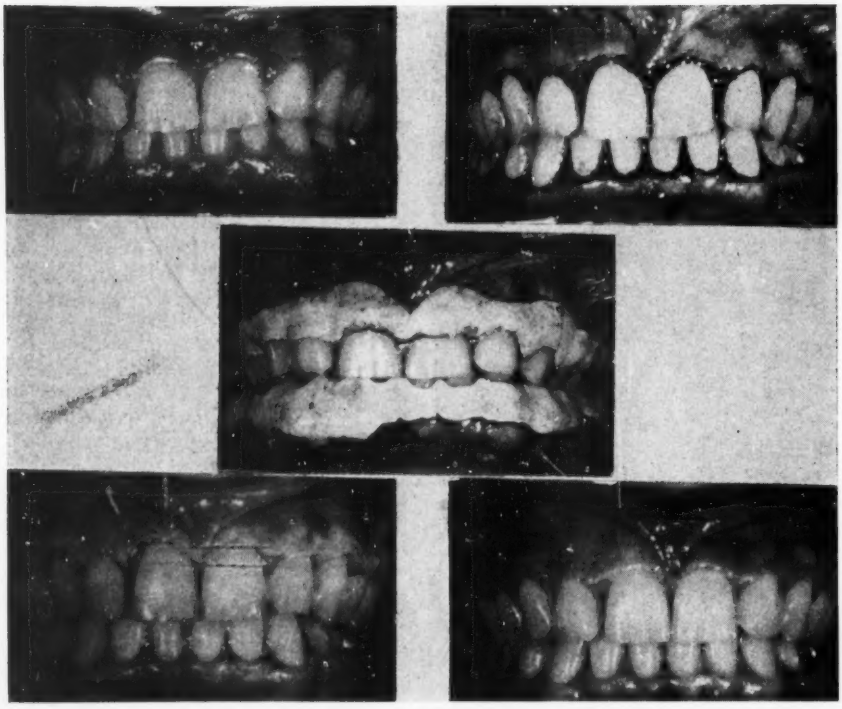

Fig. 11. Top left. Before treatment by full-mouth gingivectomy. The patient presents chronic marginal gingivitis with moderate pocket formation. Note blunting of the interproximal papillae in relation to the right mandibular anterior teeth and distortion in the contour of the interproximal papillae in relation to the left mandibular anterior teeth. Top right. Immediately after the gingivectomy using the Kirkland periodontal knives. Note that there has been no trimming or grooving of the gingiva or attempt at artificially creating gingival contours with abrasives. Center. Periodontal pack in position. Bottom left. One week after gingivectomy, immediately after pack removal. The margin of the gingiva at the line of incision is slightly rounded. Bottom right. Seven and one-half weeks after gingivectomy. The periodontal pockets are eliminated and gingival health is restored. Note the excellent gingival contour attained in the normal course of healing. In the mandibular anterior area, the previously blunted interproximal papillae on the right side are now restored and the previously distorted papillae on the left side are now well formed. The level of gingival attachment is coronal to the line of incision. (Compare with top right.)

gingivectomy, per se, did not induce recession (Fig. 8).

RECAPITULATION

The findings provide definitive information regarding clinical results obtained with a gingivectomy technic, unembellished by preliminary scaling, drugs, or so called "plastic" procedures. The unembellished gingivectomy was an effective and dependable method for eliminating the wide variety of periodontal pockets encountered in two hundred and fifty patients with periodontal disease. It is not within the scope of this study to evaluate the benefits that might be derived from attempting to enhance the unembellished gingivectomy with preliminary treatments or auxiliary "plastic" procedures. There are special conditions in which particular attention must be given to contouring the gingiva when the gingivectomy incision is made.[7] However, our findings indicate that in the attainment of gingival health following pocket eradication with the gingivectomy, it is the unimpaired healing of the gingiva which gives rise to physiologic gingival contours. Without detracting from the potential benefits afforded by embellishing

the gingivectomy with preliminary treatments or adjunctive "plastic" procedures, it must be concluded from the findings that the successful eradication of periodontal pockets and the restoration of esthetically and functionally desirable gingival contours can ordinarily be achieved without their use.

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Observations of Necrotizing Gingivitis in 870 Military Trainees*

by HAROLD E. GRUPE, D.D.S.,** AND LYNDON S. WILDER, B.S., D.M.D.,***
Camp Chaffee, Arkansas

NECROTIZING gingivitis or Vincent's infection is a continual problem in the personnel of the armed forces[1,2] and is likely to reach civilian life, since, as a rule, military service extends only through two years. Therefore, a detailed study of this disease was undertaken at Camp Chaffee, Arkansas, with the objective of circumscribing its symptomatology and with the hope of finding a method of treatment that would shorten the usual long, drawnout therapeutic procedures.[3,4]

CLINICAL FINDINGS

Necrotizing gingivitis in the age group examined (17-21 years) is not a rare disease. During the initial examination of 2,622 trainees, 58 cases, or 2.2% of those examined had Vincent's infection.

The following studies are based upon the treatment of 870 patients with Vincent's infection. The disease was, as a rule, not superimposed upon a long-standing periodontal disease, nor was it an oral manifestation of a debilitating systemic disease.[5] Instead, it was found that Vincent's infection occurs as readily in a clinically normal, clean mouth as in mouths of poor hygiene. The diagnoses were made from clinical signs and confirmed by bacterial smears[6] and biopsies.[7] The ulcerations and necrosis were sometimes confined to scattered interdental papillae; sometimes the entire marginal gingiva and occasionally, the soft palate and cheek were involved. Only three of 870 patients showed involvement of the palate and throat.

In most of the patients, the diagnosis wase made during routine examination. Subjective complaints were rare and a history of soreness and bleeding of the gingiva was elicited only after questioning. Bleeding was, in fact, the most constant sign.

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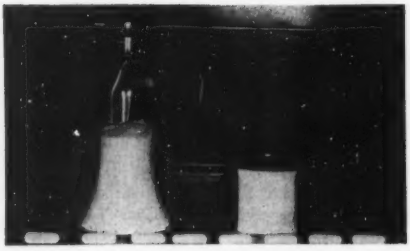

Fig. 1. Eight capsules of aureomycin powder are mixed with one sample can of denture adhesive powder (tragacanth) and used in a bottle for insufflation.

The "classical" symptoms of Vincent's infection, namely; malaise, pain, fetor ex ore, fever and lymphadenitis were found in a surprisingly small number of cases—not quite 2% of the total number. If fever was present, the temperature was rarely elevated more than a degree above normal.[8] Fetor ex ore was, if present, a late sign. That the disease is not contagious needs no confirmation.[9, 10, 11, 12]

Intramuscular injections of penicillin were dramatically effective in uncomplicated cases and were ineffective in a few "classical" cases. This seemed to confirm the interpretation of the "classical" disease as a mixed bacterial and possibly viral infection. Many of these patients gave a history of recent colds or upper respiratory infections. Frequently, herpes labialis was also present. In the extremely painful cases, the inflammation of the gingiva was much more severe and involvement of the attached gingiva and alveolar mucosa was a rather constant observation.

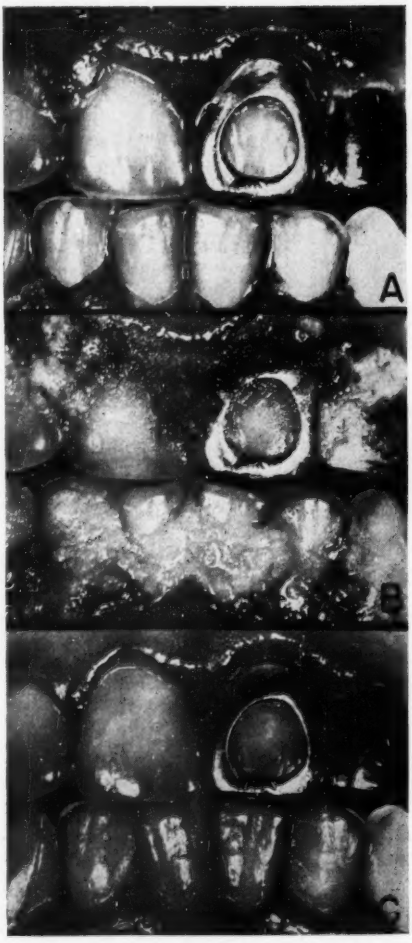

Fig. 2A. Acute necrotizing, ulcerative gingivitis —Male—Age 21, Negro—Duration 3 weeks.
Fig. 2B. Topical application of aureomycin.
Fig. 2C. Three days after aureomycin.

TREATMENT

The use of aureomycin[13] or bacitracin,[14] or the combination of penicillin with gum tragacanth[15] has been reported. In our material, the most dramatic and rapid healing was achieved by topical application of aureomycin.

A simple method of applying and holding aureomycin in contact with the tissue is as follows: An insufflator is filled with a mixture of aureomycin and denture adhesive powder—250 mg. aureomycin to 350 mg. adhesive powder (Fig. 1). This mixture is blown about the teeth and the gingival tissues. The patient is instructed not to brush his teeth. No other treatment is attempted. The patient is instructed to report back in 72 hours. In all cases which were treated in this manner, the acute destructive process was checked; dramatic results were produced with one or two ap-

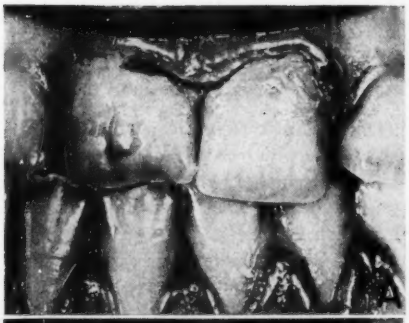

Fig. 3A. Acute necrotizing ulcerative gingivitis —one week duration—21 year old white male.

Fig. 3B. Two applications of aureomycin (three days apart). Thirty days postoperatively.

plications (Figs. 2 & 3). The patient would volunteer the information of how good his mouth felt, although he had not previously reported any discomfort. A striking observation was of the cleanliness of the teeth and oral tissues. There was a marked reduction of materia alba; often the mouth had the appearance of having been thoroughly brushed (Fig. 4).

At a second appointment, calculus may be removed if necessary. It was shown, however, that no instrumentation or homecare is necessary to insure a cessation of the acute attack. No clinical deformity of the gingival margin or papillae remains in the acute cases of initial infection unless previous periodontal disease or recession existed prior to the acute attack.[16]

MICROSCOPIC FINDINGS

Biopsy specimens from ulcerative gingivitis have rarely been reported in the literature (Box,[17] Gottlieb & Orban,[18] Schaffer[7]). Complete necrosis of the epithelium and part of the lamina propria with a fibrinous pseudomembrane on the surface, invaded by poly-morphonuclear leukocytes, is the characteristic picture of a fully developed case (Fig. 5A (F-900). The pseudomembrane consists of a network of fibrin fibers with leukocytes and often erythrocytes in the meshes of the mass (Fig. 5B (F-900). In such cases, the necrosis and ulceration are easily recognized and there are often subjective symptoms of pain present.

Fig. 4. Postoperative appearance of gingiva and teeth twenty days after two treatments with topical applications of aureomycin. Complete clinical healing and absence of materia alba. Exposure of subgingival calculus.

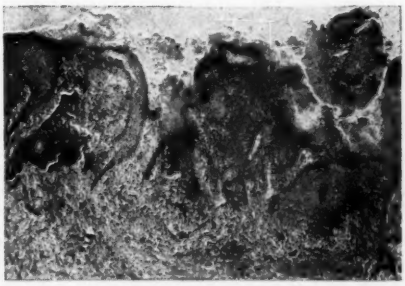

Fig. 5A. Acute ulcerative, necrotizing gingivitis (same patient as Fig. 2) of three weeks' duration. Epithelium and parts of the connective tissue necrotic.

However, in the very early stages, no

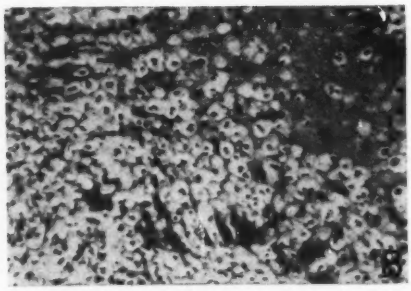

Fig. 5B. Leukocytic infiltration of the necrotic tissue and fibrinous network.

such necrotic masses are present and an ulcer is often only recognizable with a magnifying loop (Fig. 6A (F-895). The epithelium is interrupted, but the defect is covered by a fibrinous exudate, making the clinical detection rather difficult. The epithelium becomes extremely thin, but often proliferates into the inflamed connective tissue with wide and long extensions. Polymorphonuclear leukocytes invade the exudate. The underlying connective tissue is hyperemic; the capillaries dilated; the tissue edematous. The epithelium shows also numerous vacuoles in which leukocytes often can be found.

In the deeper connective tissue, masses of plasma cells are in evidence as a sign of a chronic inflammatory reaction. This chronic gingivitis in the deeper layers is a constant feature of all biopsy specimens of our cases. Between the plasma cells, large areas of tissue necrosis often appear (Fig. 6B (F-895), identical with that described by Ray and Orban.[19] The plasma cells are degenerated and round masses of necrotic material are between the cells.

If treatment is instituted, the necrosis and ulceration disappear rather rapidly. Six days after one application of aureomycin, no gross ulceration or necrosis is recognized (Fig. 7 (F-903). However, the epithelium is rather thin, edematous, invaded by leukocytes. The cells are shedding on the surface; there is no trace of a keratinous layer. In this biospy specimen, at the labial aspect of the papilla, a rather large intraepithelial microabscess can be recognized (Fig. 7B (F-903). Such an abscess can easily rupture and lead to renewed ulceration.

One month after treatment with aureomycin, the microscopic picture does not show too much improvement (Fig. 8-F-908). No ulceration or pseudomembrane formation is present. The entire surface is covered by epithelium, but the epithelium is thin; long, thin processes extend into the underlying inflamed connective tissue. The epithelium is edematous (spongiosis), invaded by leukocytes and shedding vital non-keratinized cells on the surface,—(Fig. 8B (F-908). The inflamed connective tissue is infiltrated by plasma cells with large masses of necrotic tissue between the plasma cells. The plasma cells appear degenerated in some instances.

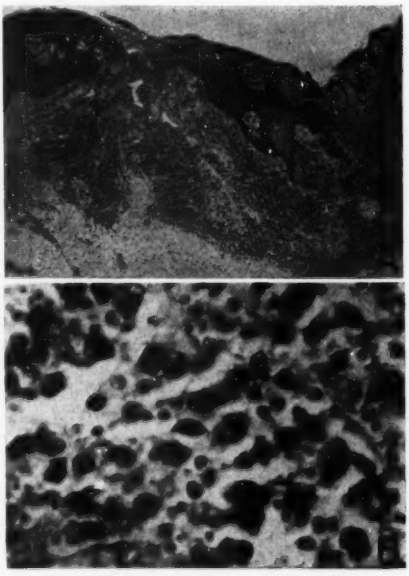

Fig. 6A. Early ulcer of necrotising gingivitis (3-4 days' duration). The epithelium is interrupted, but covered by a fibrinous exudate. Large masses of subepithelial plasma cell infiltration.

Fig. 6B. Degenerating plasma cells surrounded by round globules of necrotic tissue masses.

DISCUSSION

The most important and rather surprising finding of this study was the lack of almost all signs and symptoms of the classical picture of acute, necrotizing gingivitis in most patients. The destructive process, starting at the interdental papilla, progresses without subjective symptoms that usually bring the patient into the dental office. The conclusion, therefore, seemed inevitable that the "Vincent's infection" as usually seen and described is an advanced stage of the disease complicated by a mixed infection. The primary, uncomplicated disease is the spirochete-fusiform infection, whereas the "classical" case seems to be complicated by a secondary infection and probably viral invasion. The effectiveness of penicillin therapy in the uncomplicated; its ineffectiveness in the classical cases, is strong evidence for this interpretation. The terms, "simplex" and "complex" necrotizing gingivitis are suggested to differentiate the two diseases.

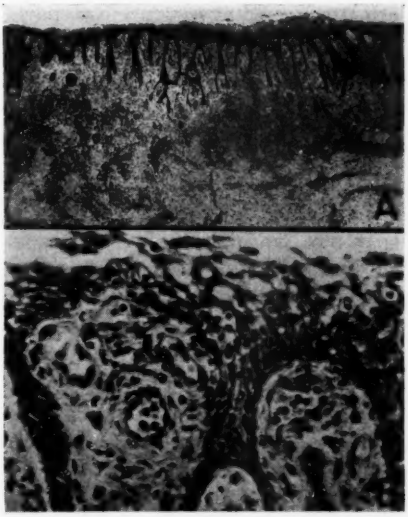

Fig. 8A. Acute ulcerative, necrotizing gingivitis one month after application of aureomycin. No ulceration or pseudomembrane present. Epithelium thin, shows long, narrow extensions into inflamed connective tissue. Plasma cells with necrotic areas in deeper regions.

Fig. 8B. Higher magnification of epithelium from Fig. 8A. Epithelium edematous; the surface shows shedding of vital cells. Hyperemia, leukocytic infiltration.

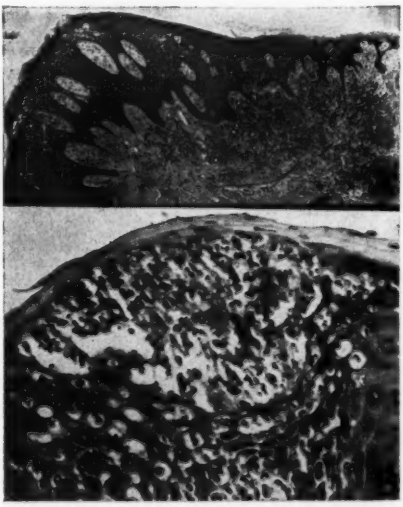

Fig. 7A. Acute ulcerative, necrotizing gingivitis six days after one topical application of aureomycin (A.R.—age 17—male). No ulceration is present, but the epithelium is extremely thin in areas of former ulceration (right upper corner of picture). Considerable plasma cell infiltration.

Fig. 7B. Microabscess in the epithelium, filled with fibrin and leukocytes. On the surface, the abscess is only covered by a few layers of parakeratotic cells. (This is a higher magnification from Fig. 7A—left side of picture.)

Treated cases of uncomplicated necrotizing gingivitis are characterized by spontaneous remissions followed by repeated new attacks. It is easily managed by local aureomycin application. However, microscopic examination of biopsy material showed that no complete healing was established. The lack of total repair seems to leave a site of reduced resistance and an easy chance of a renewed flare-up. The microscopic specimens suggest that while the acute signs of the disease—ulceration, hemorrhage, pseudomembrane formation—, disappear, no complete healing takes place. It might be necessary to follow up the treatment of the local application of aureomycin with tissue stimulation to induce the thickening of the epithelium, establish keratinization and to stimulate circulation in the connective tissue,[20, 21] thus removing the plasma cell infiltration and eliminating the necrotic tissue masses. The presence of

such large masses of necrotic tissue might be the sign of some toxic symptoms in the more extensive cases of necrotizing, ulcerative gingivitis.

The use of mild astringents, such as zinc chloride, trichloroacetic acid or silver nitrate might be indicated. The use of Superoxol might stimulate satisfactorily in eliminating plasma cell infiltration as well as increase epithelial proliferation and keratinization.[22] Further studies with biopsies will be necessary to come to a satisfactory conclusion on this question.

A word of caution should be introduced with regard to the use of aureomycin. In several cases so treated, the organism, Candida Albicans, could be grown from swabs of the cheek and tongue regions where flaky, white patches appeared. The material obtained was cultured on Sabourands Dextrose Agar and Eosin Methylene Blue. In eight days, the following findings were reported by the bacteriology laboratory—"On Sabourands Dextrose Agar, there appeared medium-sized, creamy, moist colonies with a yeast-like odor. On Eosin Methylene Blue, there appeared small star-shaped colonies that seemed to penetrate into the interior of the media. The tops of the colonies were slightly raised and acquired a bluish purple appearance. These colonies were then subcultured on fresh Sabourands Dextrose Agar, Eosin Methylene Blue and fermentation tubes containing glucose, maltose, sucrose and lactose. Whereas glucose, maltose and sucrose were fermented, lactose was not changed. Corn meal agar was also inoculated and after six days, there was seen by low-power objective, focusing directly on the plate, branching, tree-like mycelium with chlamdospores which is characteristic of Candida Albicans" (Fig. 9).

In most instances, one application of the aureomycin powder was enough to alleviate the acute signs and symptoms. If application needs to be repeated, close observation for possible monilla infection should be effected.

The primary cause of the simple necrotizing gingivitis must create conditions that allow the ever-present fusiform-spirochetal flora to become pathogenic. The effect of mental stresses has already been adduced as an explanation.[23] The fact that the simple form was found in 2.2% of recent inductees, speaks for the probability of a psychosomatic factor. The coincidence of the necrotizing gingivitis with nail biting in numerous cases, adds more strength to this assumption. Stressor effect on arterioles of the oral vascular bed could manifest itself first in the papillary tissues since they are mainly supplied by the terminal branches of the septal or interdental arteries with scant anastomoses with vestibular or lingual gingival arteries, thus almost resembling end arteries.[24]

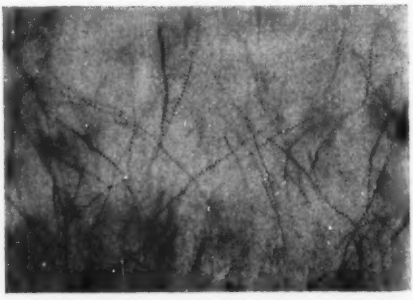

Fig. 9. Low power photomicrogram of Candida Albicans growing on potato agar.

CONCLUSIONS

1. Acute necrotizing, ulcerate gingivitis occurred in 2% of newly inducted army personnel in the age group of 17-21 years.

2. Few of the men presenting an acute, necrotizing, ulcerative gingivitis show any subjective symptoms. The diagnosis is made at routine examination.

3. The signs are bleeding of the gingiva and typical ulcers, especially at the tip of papillae.

4. In cases showing the "classical" signs

and symptoms of Vincent's infection, a viral infection seems to be superimposed upon the fusiform-spirochetal infection.

5. A psychosomatic factor seems likely as the predisposing agent, acting primarily on the papillary arterioles that resemble end arteries.

6. Local applications of aureomycin in gum tragacanth leads to clinical healing. Biopsies, however, reveal a lack of complete epithelial repair.

7. Follow-up treatment of tissue stimulation is suggested to induce complete healing and prevent recurrences.

Appreciation is expressed to the Colorado Dental Foundation for the microscopic specimens and the photomicrograms.

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Current Knowledge of Nutrition Relative to the Supporting Dental Structures

by SAMUEL DREIZEN, D.D.S.,* *Chicago, Illinois*

THE vitamins are chemical compounds with definitive physical and chemical properties (Fig. 1). So specific is their molecular arrangement that the substitution of one or more atoms may convert nutritionally essential vitamins into nutritionally deleterious antimetabolites. Biochemically, most, if not all vitamins are irreplaceable components of enzyme and coenzyme complexes. As such, they may be regarded as organic catalysts in the chain of chemical reactions which occur continuously at the cell level. During these reactions the vitamin-containing enzymes undergo cleavage and although the process is ostensibly reversible, re-synthesis is frequently incomplete. This, together with relative lack of body storage, leads to the need for constant vitamin replacement. Failure of vitamin supplies to meet tissue demands is followed by the development of "biochemical lesions" which, if persistent and uncorrected, are ultimately productive of tissue breakdown.

The transition from the "biochemical lesion" to the clinically apparent anatomic lesion is essentially a degenerative process stemming from the sustained interruption of vital cellular activities. The tissue breakdown is accelerated, intensified and compounded by secondary factors such as infection and trauma. The secondary factors undoubtedly contribute to the predilection for the lesions to develop selectively in particular anatomic sites. The combination of traumatic and infective forces operating simultaneously in the oral cavity renders this area exquisitely sensitive to the pathologic changes which reflect the deficiency states. Paradoxically, however, the underlying pathology so specific in causation is, more often than not, non specific in its overt manifestations. The so-called classic case of deficiency disease is in fact the rare case; the so-called atypical case is the usual case. When present, classic signs are almost always associated with the late stages of the disease. Thus, unlike the uniformity obtainable in completely controlled animal experiments, naturally occurring human deficiency syndromes are characterized clinically by an individuality of response.

Despite the variability in changes of nutritional origin which affect the oral cavity, excellent diagnostic opportunities are available. The diversity of tissues among the supporting structures of the teeth, their early involvement in nutritional disorders, and their accessibility to physical and roentgenographic examination facilitate recognition of the deficiency states. Early recognition is the key to successful treatment. There are no laboratory tests for the deficiency syndromes which are absolutely pathognomonic. The diagnosis of nutritional dyscracias is primarily a clinical problem requiring a reliable history, a careful and complete physical examination and a working knowledge of tissue reactions to prolonged deprivation of essential chemical substances. A response to proper and persistent therapy is empiric proof of accuracy of diagnosis.

Common features in the etiology of the deficiency diseases favor the development of multiple rather than single syndromes. Variations in the intensity of, and tissue propensity to each nutritional deficiency may complicate and confuse the clinical picture. A lack of nicotinic acid can mask the symptomatology of a concurrent riboflavin inadequacy. The latter may, in turn, suppress the manifestations of pyridoxine deficiency. In treating each case, it is necessary to delineate the lesions in terms of each of the nutrients which contribute to the maintenance of the integrity of the involved tissues. For ease of presentation the

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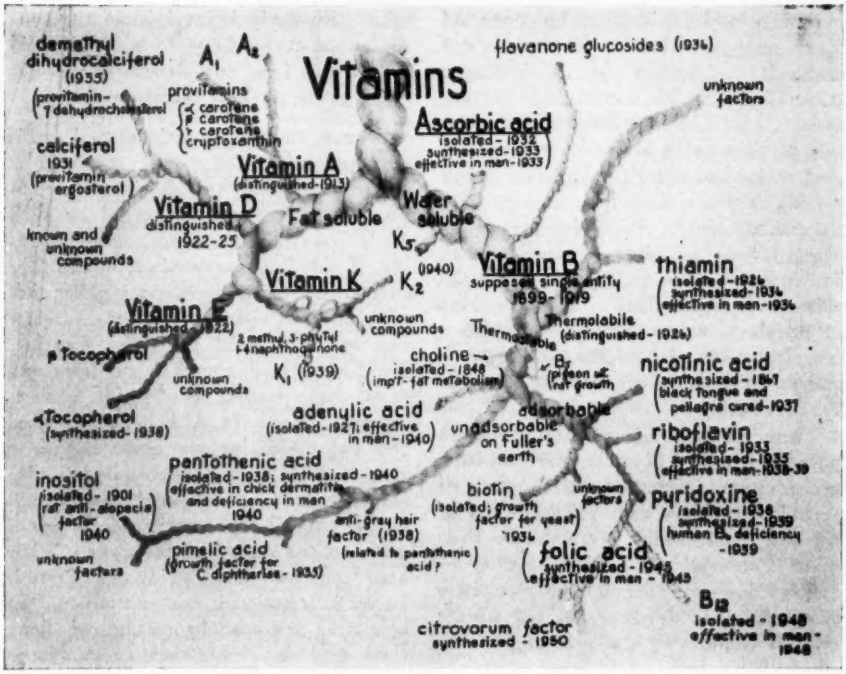

Fig. 1

manner whereby each deficiency state may affect the supporting dental structures and related tissues will be treated separately. Their possible coexistence must, however, always be borne in mind.

Vitamin A: Vitamin A is a fat soluble high molecular weight alcohol attached to a β-ionone ring. Its fundamental role in metabolism is unknown. The presence of five unsaturated double bonds in its molecule suggests a probable function as an oxidation-reduction catalyst. In man, the deficiency is rarely seen in the absence of diseases which interfere with the digestion and absorption of fat, or which prevent the conversion of carotenoid precursors into Vitamin A.

The basic pathology in vitamin A deficiency is the replacement of certain specialized epithelium by a stratified squamous keratinizing epithelium[1]. Some epithelial structures are damaged earlier than others. In man, the oral epithelium is involved late, with the reported manifestations being hyperplasia and hyperkeratosis of the gingivae, leucoplakia and hypoplastic changes in the enamel and dentin due to severe damage to the developing enamel organ[2,3,4]. The keratinized epithelium is presumably very susceptible to bacterial invasion and in the late stages of this disease severe secondary infections of the oral cavity may occur. The gingival and mucosal lesions attributable to a vitamin A deficiency are clinically identical with those caused by a variety of local and systemic conditions. Because of the rarity of vitamin A deficiency, a diagnosis based exclusively on the presence of oral lesions is untenable in the absence of deficiency signs and symptoms in other parts of the body.

Vitamin B Group:

Thiamin: Thiamin (vitamin B_1) is a water soluble compound composed of a

pyrimidine and a thiazole group connected by a methylene bridge. The methylene bridge is split by the enzyme thiaminase present in many fish and molluscs. As cocarboxylase (thiamin pyrophosphate), thiamin participates in oxidative decarboxylations which lead to the formation of carbon dioxide. The major symptomatology of a thiamin deficiency is associated with its action as an inhibitor of cholinesterase formation at nerve endings. In prolonged thiamin depletion there is a degeneration of peripheral nerve fibers extending to a complete destruction of the myelin sheath and axis cylinder with ultimate death of the nerve cell.

There are no suggestive oral lesions in pure vitamin B_1 deficiency. Patients with a generalized deficiency of the B group of vitamins may show a marked sensitivity of the oral tissues which is relieved by thiamin.[5] Conversely, the administration of massive doses of thiamin may precipitate symptoms of a deficiency of one of the other B vitamins such as nicotinic acid or riboflavin.[6] There are indications that a physiological relationship may exist between thiamin and ascorbic acid. Erosive necrotic lesions beginning around the teeth and extending almost entirely around the lower jaw have been produced experimentally in thiamin deficient dogs. The lesions promptly healed following the administration of vitamin C, a non essential nutrient for the dog. These findings suggest that an absence of thiamin interferes with the synthesis of vitamin C in this animal.[7] In man, it has been reported that the onset of scurvy on a minimal intake of vitamin C is delayed by small amounts of thiamin and that the antineuritic action of thiamin is increased by ascorbic acid.[8]

Riboflavin: Chemically, the riboflavin (vitamin B_2) molecule consists of an iso-alloxazine nucleus with a ribityl side chain attached to the middle ring. The iso-alloxazine portion is capable of alternate oxidation and reduction, a property which enables riboflavin to participate in a number of enzyme systems concerned with cellular respiration. Together with pyridoxine, riboflavin is involved in the conversion of tryptophane to nicotinic acid and as such is of considerable importance in the pathogenesis of endemic pellagra.

In man, the most prominent oral manifestations of ariboflavinosis are angular stomatitis, cheilosis and glossitis. Their appearance is frequently preceded by such prodromal symptoms as soreness and burning of the lips, tongue and mouth. The angular stomatitis begins as a pallor followed in short order by erythema, maceration and superficial transverse fissuring. The fissures may extend up or down and cross the vermilion border for a short distance. They have red bases, rarely bleed and eventually become covered with a honey-colored crust. Unless secondarily infected by bacteria and fungi, they are not unduly painful. The severity of the lesions may wax and wane with the seasons. While both angles are usually involved, a unilateral distribution is not uncommon. The lips often become abnormally red along the line of closure and show an increase in vertical fissuring due to superficial denudation of the mucosa.

The glossitis associated with ariboflavinosis is marked by changes in color, topography and sensitivity. In advanced cases, the tongue may take on a purplish red or magenta hue due to a dilatation and proliferation of the capillaries with a slowing down of the circulation. The papillae become mushroom-shaped with the overlying epithelium flattened and swollen to give the dorsum a pebbly or granular appearance. The tongue may be painful and burn when food is eaten.

Evidence derived from animal studies, but as yet unconfirmed in the human, indicates that a deficiency of riboflavin per se or in concert with other members of the B group may affect both the hard and soft supporting structures of the teeth. Congenital dentofacial anomalies most striking in the lower jaw and palate have been produced in rats by maternal riboflavin deficiency.[9] Lesions ranging from a mild gingivitis to necrotic ulcerative gingivitis

and noma have been produced in monkeys maintained on a vitamin B_2 complex deficiency diet.[10]

Nicotinic Acid: Niacin, nicotinic acid and 3-pyridine carboxylic acid are synonyms for a vitamin which forms part of complex enzyme systems concerned with hydrogen transport in living cells. As the functional group of coenzyme I (diphosphopyridine nucleotide) and coenzyme II (triphosphopyridine nucleotide), nicotinic acid plays an important role in carbohydrate metabolism. In addition to being ingested with food and synthesized by intestinal bacteria, part of the body's requirements for niacin is provided by the transformation of tryptophane to nicotinic acid in the tissues. Ten to twenty parts of l-tryptophane are needed to form one part of niacin. The transformation from tryptophane proceeds first to indoleacetic acid and kynurenine. Kynurenine is then converted to 3-hydroxyanthranilic acid which is metabolized through quinolinic acid to niacin. Riboflavin is required for the formation of 3-hydroxykynurenine from kynurenine, and pyridoxine is needed for the conversion of the latter to 3-hydroxyanthranilic acid. Thus, inadequacies of tryptophane, riboflavin and pyridoxine may all contribute to a niacin deficiency.

The oral symptoms of pellagra are among the first to appear and in many cases constitute the presenting complaint. Anorexia, burning of the tongue and abdominal pain which feature the prodromal period gradually give way to an intense glossitis, stomatitis, gingivitis, pharyngitis, gastritis and enteritis. The lips become reddened, ulcerated, scaly and cracks may appear at the corners of the mouth. The tongue becomes swollen, indented by the pressure of the surrounding teeth and fiery red. The redness begins at the tip and margins and subsequently spreads to involve the entire dorsum. The lingual papillae undergo atrophy accompanied by desquamation of the overlying epithelium. As desquamation progresses the tongue becomes smooth, dry and beefy red in color. The tongue is extremely painful and often develops superficial ulcerations. The gingivae are reddened, ulcerated and denuded and the pharynx may have a similar appearance. Vincent's infection and Vincent's angina are frequently superimposed on the devitalized tissue. Microscopically, the oral lesions resemble those of the skin with congestion of the papillary vessels, edema of the papillae and moderate lymphocytic infiltration of the submucosa.

King[11] has postulated an etiological relationship between niacin deficiency and Vincent's infection. While some investigators have used nicotinic acid successfully in the treatment of ulceromembranous gingivitis, others have failed to obtain beneficial effects.[12, 13, 14, 15] The disparity in results indicates that niacin is not a contributing factor in all cases of Vincent's infection. When it is, correction of the deficiency followed by a restoration of the vitality of the affected tissues leads to a subsidence of the secondary infection. Clinically, this is analagous to secondary invasion of the angular stomatitis of ariboflavinosis by hemolytic *Staphylococcus aureus* and *Streptococcus hemolyticus* and their disappearance following healing of the lesions with riboflavin therapy.[16]

Folic Acid: The folic acid molecule consists of an amino acid residue (glutamic acid) attached to a B vitamin residue (para-aminobenzoic acid) linked in turn to a pteridine group. The entire structure constitutes pteroylglutamic acid. Folic acid is present in foods partly as the free form but mainly as the conjugate which contains several glutamic acid groups. Both forms are transformed into biologically active tetrahydroformyl folic acid (folinic acid) probably within the liver. It is believed that ascorbic acid is required for this transformation. Biologically, folic acid is essential for nucleic acid and nucleoprotein synthesis, thereby being required by all living cells. Its absence leads to a megaloblastic arrest in the bone marrow which characterizes the macrocytic anemia of sprue, nutritional macrocytic anemia, megaloblastic anemia

of infancy, macrocytic anemia of pregnancy and pernicious anemia.

In the monkey the typical oral lesions of a pure folic acid deficiency are gingivitis and necrosis of the oral and gingival mucosa.[17] In man, the syndromes which respond to folic acid all have the common findings of glossitis, stomatitis and macrocytic anemia. In the early stages, the tongue is inflamed with crops of minute whitish painful ulcerations demarcated by fiery red borders. In the advanced stages there is total atrophy of the filiform and fungiform papillae. Inflamed hypersensitive gingivae and apthous stomatitis frequently accompany the tongue changes. Soreness and burning of the tongue is a common presenting complaint in these patients. Recently, Ross[18] described a case of sprue with advanced periodontal destruction which was attributed to the impaired absorption of the vitamins and minerals concerned with bone formation. Such findings exemplify the cyclic relationship of the deficiency states wherein the organic disturbances created by a lack of one nutritional essential lead to defects in the ingestion, absorption and assimilation of other required nutrients.

Vitamin B_{12}: The molecular configuration of vitamin B_{12} has recently been established by Todd and associates.[19] It is the heaviest and most complicated non protein material essential for human nutrition with the empirical formula of $C_{63}H_{90}O_{14}N_{14}PCo$. Benziminazole nucleotide is an integral part of the nucleus which constitutes the basis for a whole family of substances with similar hemopoietic activity. Vitamin B_{12a} and vitamin B_{12b} are identical. Vitamin B_{12} differs from vitamin B_{12a} (vitamin B_{12b}) in containing a cyanide group coordinated with the cobalt atom. The terms cyanocobalamin and hydroxycobalamin have been applied appropriately to vitamin B_{12} and vitamin B_{12a} (vitamin B_{12b}) respectively. Like folic acid, vitamin B_{12} is essential for megaloblastic erythropoiesis to a normoblastic type. Unlike folic acid it prevents the onset of subacute combined degeneration of the spinal cord in pernicious anemia. To be absorbed effectively from the intestinal tract, vitamin B_{12} must be potentiated by the intrinsic factor contained in normal gastric juice.

In man, the most severe form of a vitamin B_{12} deficiency is pernicious anemia. Other macrocytic anemias are thought to be mild forms of vitamin B_{12} deficiency complicated by varying degrees of folic acid deficiency. Approximately 60 per cent of all patients with pernicious anemia have recurrent attacks of glossodynia which may be the initial manifestation but which usually appears after the disease is well established. The soreness may last for several weeks after which it disappears spontaneously only to recur. During the exacerbations the tongue is extremely painful and fiery red. The entire dorsum is usually involved, but it may be confined to well circumscribed areas. Occasionally there are associated superficial whitish ulcerations. In about 50 per cent of the cases there is atrophy of the lingual papillae which commonly progresses to complete disappearance leaving a smooth tongue. In cases which progress to subacute combined degeneration of the spinal cord, fiery red, excruciatingly painful mucous membrane lesions develop which involve the gingivae, buccal mucosa and angles of the mouth. They are not responsive to folic acid but clear completely following the administration of vitamin B_{12}.[20]

Pyridoxine: Like nicotinic acid, pyridoxine is a pyrimidine derivative. As codecarboxylase (phosphorylated pyridoxine) it is the prosthetic group of enzymes involved in the removal of carbon dioxide from many amino acids and the transfer of amino groups from one compound to another. As mentioned previously it is also concerned in the conversion of tryptophane to niacin.

Although a deficiency of pyridoxine has been shown to produce degenerative changes in the periodontal structures of mice,[21] little is known of the effects of an absence of this vitamin on the supporting struc-

tures of human teeth. The clinical manifestations of human pyridoxine deficiency were recently elucidated by Mueller and Vilter[22] who succeeded in producing a deficiency state in subjects maintained on a vitamin B poor diet containing added desoxypyridoxine, an antimetabolite of pyridoxine. The resulting syndrome included seborrhea-like skin lesions about the mouth with erosions of the angles which resembled ariboflavinosis. Some subjects developed sore, swollen red tongues and lesions of the oral mucous membranes which were similar to those of a nicotinic acid deficiency. The lesions did not respond to riboflavin, nicotinic acid or thiamin but healed within three days after administering pyridoxine.

Pantothenic Acid: Pantothenic acid forms the prosthetic group of coenzyme A, a compound involved in the transfer of acetyl groups in carbohydrate and fat metabolism. Evidence is accumulating that this vitamin may be concerned also with adrenal cortical function. As yet, however, neither an experimentally induced nor a naturally occurring pantothenic acid deficiency has been described in the human. Isolated reports indicate that pantothenic acid may be effective in the treatment of glossitis and cheilosis resistant to other members of the B group of vitamins.[23,24]

Vitamin C: The chemical characteristic distinctive of ascorbic acid is the presence of a dienol group in the molecule which contributes to its reducing action and confers acidic properties on the compound. Vitamin C is the least stable of the vitamins being extremely sensitive to alkalies and oxidation particularly in the presence of iron and copper ions. Dehydroascorbic acid, the first oxidation product of vitamin C is a potent antiscorbutic agent which in man can replace ascorbic acid on a weight for weight basis. Physiologically, ascorbic acid acts as an hydrogen transporter in tyrosine metabolism, seems to be involved in the production of adrenal cortical hormones, plays a part in erythropoiesis and is essential for connective tissue formation and wound healing.

Gingivitis is one of the most reliable early signs of clinical scurvy. The gingivae are engorged, discolored, boggy and tender. The discoloration ranges from bright red through bluish purple to black. The gingival tissues bleed readily in response to slight pressure. The dental papillae are usually hypertrophied. With time, the teeth become mobile due to the imbalance between the respective rates of degeneration and regeneration of the periodontal membrane and to the resorption of the alveolar bone. If untreated, the teeth may exfoliate as a result of the complete destruction of the periodontium. In addition to the gingival hemorrhages and hyperemia there is epithelial degeneration and ulceration with superimposed microbial infection. The gingival symptoms of scurvy do not develop in the absence of teeth. In infants and young children the lesions may be confined to the areas directly over the erupting teeth. The involved mucosa becomes spongy, swollen and bluish in color. Following eruption the teeth may be partially or completely covered by hypertrophied gingivae. In patients with a partial dentition, the scorbutic lesions are restricted to the gingival areas immediately surrounding the teeth, the edentulous areas being symptom free.

Except for its undeniable therapeutic value in demonstrable scurvy, clinical studies dealing with the role of vitamin C in maintaining the health of the supporting structures of the teeth have been inconclusive and somewhat contradictory. Much of the confusion stems from failure to differentiate between scorbutic gingivitis and the gingivitis which may mimic scurvy clinically. Furthermore, there are indications that scurvy may be due to a combined deficiency of ascorbic acid and vitamin P, a dietary factor essential for the maintenance of capillary resistance in the human.[25,26] Recent findings do not support the concept that ascorbic acid deficiency per se results in increased capillary fragility and no important changes in blood vessels have been described which can be attributed to this deficiency.[27,28] Evidence

has been offered that in scurvy there is a failure of the contractile mechanism of the small blood vessels leading to dilatation and a sluggish blood flow which could account for the engorgement and discoloration of the gingival tissues.[29]

Vitamin D: There are at least sixteen different compounds related to the sterols which have antirachitic properties. They vary widely in potency and show marked species differences. The most important of these are vitamin D_2 (calciferol) produced synthetically by the irradiation of ergosterol and vitamin D_3 (activated 7-dehydrocholesterol) formed naturally in the skin by the action of ultraviolet light on the sterol precursor. In general, vitamin D_2 and vitamin D_3 have the same biological effects, notably the activation of alkaline phosphatase, promotion of the absorption of calcium and phosphorus from the intestinal tract and regulation of the resorption of phosphates from the kidneys.

While the skeletal changes in rickets are most pronounced in those regions of the body where growth is most rapid, alterations in the supporting structures of the teeth have been noted in experimental animals on vitamin D deficient diets.[30,31] The primary pathology in the alveolar bone is failure of the newly formed osteoid tissue to undergo mineralization and osteoclastic resorption. The usually thin osteoid border becomes very wide and compresses the periodontal tissues and blood vessels leading to hyaline degeneration of the connective tissue and in some instances to complete obliteration of the periodontal membrane. In the adult form of human vitamin D deficiency (osteomalacia) absence of a lamina dura about the teeth is a characteristic roentgenologic finding. The lack of a lamina dura helps to differentiate osteomalacia from osteoporosis, a disorder of protein metabolism wherein there is a decrease of bone mass resulting from a decrease in bone matrix formation.

Vitamin K: Vitamin K is a group name applied to quinone compounds with antihemorrhagic properties. Both natural (vitamin K_1 and vitamin K_2) and synthetic forms (vitamins K_3, K_4, K_5, and K_6) are now available. Vitamin K_1 is present in foods and vitamin K_2 is formed by the bacterial flora of the intestinal tract. Because of the latter, and because bile salts are required for the absorption of naturally occurring K vitamins, the hemorrhagic tendency induced solely by a lack of vitamin K rarely, if ever, occurs in man in the absence of hepatic, biliary tract or intestinal disease. Patients on prolonged dicoumarol therapy may possibly develop a vitamin K deficiency as dicoumarol is a competitive inhibitor of this vitamin.

Mild cases of vitamin K deficiency show gingival bleeding on vigorous brushing of the teeth; severe cases show a slow oozing of blood from the gingivae similar to that noted in other diseases which disturb the blood clotting mechanism.[32] In vitamin K deficiency, coagulation time is prolonged, capillary fragility is unaltered and prothrombin time is increased.

The contributions of the aforementioned vitamins to the maintenance of the normal structure of the oral tissues is essentially indirect, affecting as they do metabolic processes within cells rather than constituting an integral part of tissue structure per se. In contrast, deficiencies of calcium, phosphorus and protein directly affect the structural composition of the periodontium. In animals, experimentally induced pure calcium and phosphorus deficiency results in an excessive resorption of alveolar bone and osteoporosis;[31] in pure protein deficiency there is degeneration of the connective tissue of the gingivae and periodontal membrane, osteoporosis of the alveolar bone and retardation in the deposition of cementum.[33] In man, deficiencies of the calcifying minerals and disturbances in protein metabolism contribute to the development of osteomalacia and osteoporosis respectively, in each of which there are detectable changes in the structure of the alveolar bone. The precise effects of pure deficiencies of calcium and/or phosphorus and of single or multiple essential

amino acids on the supporting dental structures in the human remain to be determined.

CONCLUSION

The supporting dental structures are sensitive to deficiencies of many of the nutrients which are essential for man. In health, these tissues are admirably suited to withstand the stresses to which they may be subjected. In the depleted state, repeated exposure to chemical, mechanical and microbial irritants contribute to their rapid breakdown. The ensuing lesions may be localized or generalized in distribution and acute or chronic in course. They are highly individualistic in pattern and rarely take the identical form in different patients. They frequently have a multiple rather than a single causation. Deficiencies of borderline intensity or of short duration may occur without the appearance of some or all of the characteristic signs. Conversely, the presence of a "deficiency" sign need not connote a deficiency state since many are non specific in origin. In the diagnosis of these conditions, dietary histories, physical examination and laboratory tests are contributory; response to the specific suspected factor is confirmatory.

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Periodontia and the Medical Profession

by RAOUL H. BLANQUIE, D.D.S., *San Francisco, Calif.*

SEVERAL decades past, the focal infection theory attained its height of popularity following revealing findings from the published reports of investigators such as Billings, Rosenow, Haden and others. With the advent of these startling revelations, leading to a new approach of attack upon diseases of infectious or doubtful origin, dentistry found itself sharing the burden of blame for many of these ailments. Then followed an era when the popular prescription, for the treatment of many systemic disorders, was the indiscriminate removal of teeth and in particular the non-vital ones. Periodontal infections at that time, were generally bypassed unless their progress was so far advanced as to make extractions mandatory. Successful systemic results followed in certain cases, after the extraction of suspected teeth, while in others disappointment prevailed.

With the passing of years the focal infection theory has not been discarded, nor lost its importance, but has been placed in its proper perspective in relation to other causative factors of disease. It still retains its full significance wherever it may be related. That diseased teeth and their supporting structures can be primary or secondary foci of infection, capable of inaugurating or contributing to systemic disturbances, is an established fact based upon both laboratory and clinical findings. In contrast with original thought, we find today that periodontal diseases are considered the most important foci of infection in the oral cavity because they are the most prevalent and contain a heavier bacterial flora composed of more potentially pathogenic organisms.

It is therefore natural that physicians, while seeking etiological factors, examine the oral cavity for possible clues to the conditions that they are endeavoring to remedy. Such practice can only be commended and encouraged. Many physicians, upon finding or suspecting oral infection, make it a point of communicating with the patient's dentist and consulting with him

as to the possible trouble and the measures necessary for its correction. This group of medical colleagues merits the highest praise of the dental profession and should be given its fullest cooperation.

On the other hand, in many communities, will be found another type of medical men whose attitude towards dental problems has been harmful to the public dental welfare and has brought discredit to members of our profession. Physicians in this particular group rely mostly upon their own personal ideas of dental diagnosis and treatment with the result that many teeth are lost that otherwise could be retained through proper therapeutic measures. Their approach is also often not compatible with professional ethics causing many dentists to come under the pale of suspicion by their patients.

Complaints are frequently heard from dentists that physicians, without prior consultation, have ordered the extraction of teeth of their patients and sent them directly to another dentist or to an oral surgeon to carry out their requisites. Often these cases involve the loss of recently placed restorations, bridgework or partial prosthesis. When these patients are informed by their physicians that extractions are necessary, in order to relieve their systemic disturbances, doubt is reflected in their minds regarding the judgment and competence of their dentists. This absence of cooperation would clearly seem to indicate that these medical men lack faith in their dental confreres' knowledge of basic sciences and limit their appreciation of our profession solely to its mechanical phases. It is a paradoxical fact that physicians generally depend upon the radiographer's report of x-rays for diagnosis of their own cases, yet regarding dental roentgenograms they show no hesitancy to examine them personally and formulate their own interpretations.

During the initial period of popularity of the focal infection theory, the medical profession centered its attention principally upon devitalized teeth and periapical infections. Endodontia had not then attained its present development and the general belief that many of these teeth should be removed was probably not entirely without foundation. Physicians' suspicion towards advanced carious teeth may somewhat be justifiable as today we still do not possess any positive index to determine the presence or absence of infection in the living pulps of such teeth. At the present time much of the medical profession's attention is directed towards infections of the periodontium. The resulting loss of alveolar bone in any degree seems to warrant many physicians to believe that the affected teeth should be extracted in order to eliminate the infection. That systemic infection is a potential factor in all types of periodontal disturbances, even before the alveolar bone is affected, cannot be disputed. A discussion of periodontal disease with certain physicians will disclose their belief that they regard any disintegration of alveolar bone as being associated with an osteitis or an osteomyelitis.

This viewpoint is entirely without scientific background and clearly demonstrates an absence of histo-pathological knowledge. It fails to appreciate the known and accepted findings that the infectious portion of a periodontal lesion does not lie in the alveolar bone, but in the overlying soft tissues. Thus to remove a tooth in order to eliminate infective processes of a periodontal lesion, unless the condition is too far advanced and beyond repair, is as logical as would be the amputation of a finger in which infection was present. The logic of medical belief that teeth affected by periodontal disease should be extracted is based upon the premise that systemic symptoms have disappeared following their extraction. This evidence cannot be dismissed as extraction is the radical procedure of eliminating periodontal lesions which automatically disappear upon the removal of the affected teeth. The point of total disagreement, between the medical and dental viewpoint, is that the latter believes that infections of the periodontium can be completely eradicated without resorting to the

sacrifice of the teeth. This contention can be supported by laboratory and clinical evidence.

Many medical men fail completely to recognize the fact that just as endodontia today possesses wider knowledge and better techniques to accomplish its objectives, so does periodontia have similar equipment to successfully treat and eliminate periodontal infections and prolong for many years the function and life of teeth in a healthy environment without jeopardizing the welfare of the patient. When our medical colleagues are informed that positive techniques are employed to eradicate the periodontal lesion in which the infective processes are lodged, they are little impressed believing that the infection still persists in the calcified structure.

It is evident that a solution is required to remedy this unfortunate situation. If members of the dental profession are to see an end of cases sacrificed through the whims and fancies of well-meaning but misinformed physicians, certain expedients will have to be resorted to. Dentists have been trained in the basic sciences and there is no reason why they should be by-passed and ignored in the solution of dental problems. It is therefore obvious that adequate measures to bring about satisfactory cooperation between members of the medical and dental professions are urgently needed to end the unnecessary sacrifice of dental organs.

Since teeth and their appendages are integral parts of the body, it would seem that the curriculum of medical schools should include a certain amount of dental histology and histo-pathology. By this means only can physicians have the necessary knowledge towards properly evaluating the processes of dental health and disease.

In localities wherein medical and dental societies both exist, joint meetings could be arranged at which time mutual problems could be discussed. Such meetings would accomplish a great deal towards clearing the atmosphere of misunderstanding.

Another approach that would ameliorate the situation would be articles written by dentists, on the subject of dental infections and their treatment, to be published in medical journals. Dentists who are attached to hospital staffs could arrange to speak before staff meetings and enlighten their medical brethren on the pertinent subject.

The situation under discussion is not a remote one nor of unusual occurrence and is prevalent in many localities. Nevertheless it must not be construed as a blanket indictment against the entire medical profession. Many physicians do try to cooperate with dentists whenever the field of dentistry becomes involved in their diagnosis and treatment. The criticism is aimed principally at that segment of the medical profession that fails to appreciate that dentists are qualified physicians and surgeons of the oral cavity and are equipped with scientific knowledge to diagnose and correct ailments and disorders occurring in their field.

Gingival Pyogenic Granuloma

by David F. Mitchell, D.D.S., Ph.D., *Minneapolis, Minnesota**

PYOGENIC granulomata may appear anywhere on the skin or mucous membrane of the body, but are especially common on the gingivae. They are associated often with calculus, poor oral hygiene, irritating dental restorations, trauma due to dental operations, or the indiscriminate use of toothpicks.

In a review of the literature concerned with this lesion Kerr[1] found agreement among recent writers that granuloma pyogenicum usually was due to minor trauma, which allowed the invasion of microorganisms of low virulence, resulting in a characteristic tissue response. This is an overgrowth of inflamed, very vascular connective tissue (granulation tissue), which is usually covered with squamous epithelium; however, areas of ulceration of the surface frequently are seen. Thus it is not a true tumor, but rather a localized hyperplastic growth of tissue resulting from irritation. In this respect it may be compared with the overgrowth of tissue associated with continued wearing of ill fitting dentures—the so-called denture injury tumor. Or it may be compared with the piling up of tissue at the orifice of a fistula, or even with a periapical granuloma. Thus, the overzealous attempt to heal after minor trauma and/or infection produces granulation tissue.

Clinically, the lesion is a raised, red, peripheral growth, either sessile or pedunculated, which may appear in the course of a week or more and persist for years if untreated. It may be smooth or lobulated, and tends to bleed easily after manipulation. It seldom grows larger than one centimeter in diameter. Small amounts of new bone are found sometimes within the growth. It may remain static, or if the cause is removed or the physiologic state of the patient is changed it may grow smaller. In the latter case the number of blood vessels decreases, the inflammatory reaction lessens, and a mass of fibrous tissue covered with epithelium is left. This is probably the origin of many "fibromas" or fibroepithelial polyps found in the mouth.

Microscopically, the tissues of the so-called pregnancy tumor and hyperplastic gingivitis of pregnancy are identical with those of pyogenic granuloma. Presumably the hormonal changes of pregnancy predispose to the formation of pyogenic granuloma in some cases. Another predisposing factor is the existence of an hemangiomatous base from which the pyogenic granuloma may spring.

Treatment of these localized inflammatory lesions consists of the removal of any causative irritating factor that may be present, followed by complete excision of the growth and microscopic study of the tissue.

The accompanying illustrations show four cases of pyogenic granuloma of varying degrees of severity.

Case 1. A 33 year old female presented with a small lesion of the gingiva between the upper left lateral incisor and the attached pontic replacing the adjacent central. The duration was 10-12 months. There were no pertinent radiographic findings. Treatment consisted of removal of debris from beneath the pontic, excision

University of Minnesota, School of Dentistry, Photography by Dental Illustration Laboratory.

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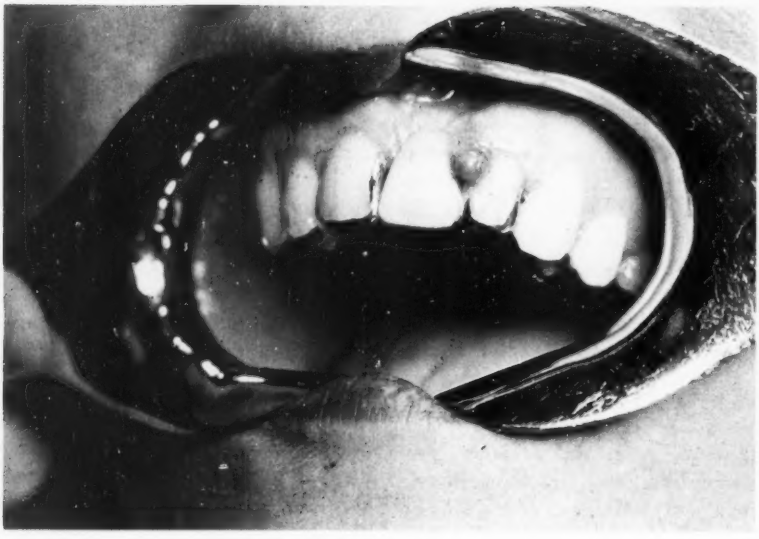
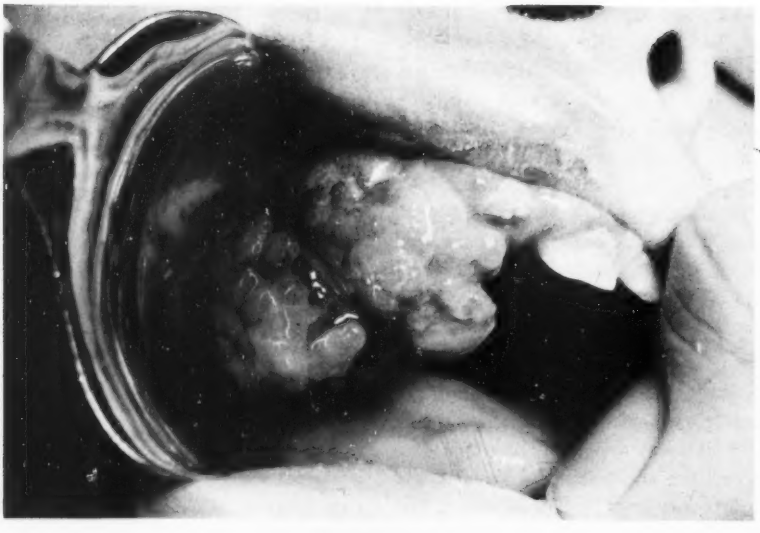


Case 1



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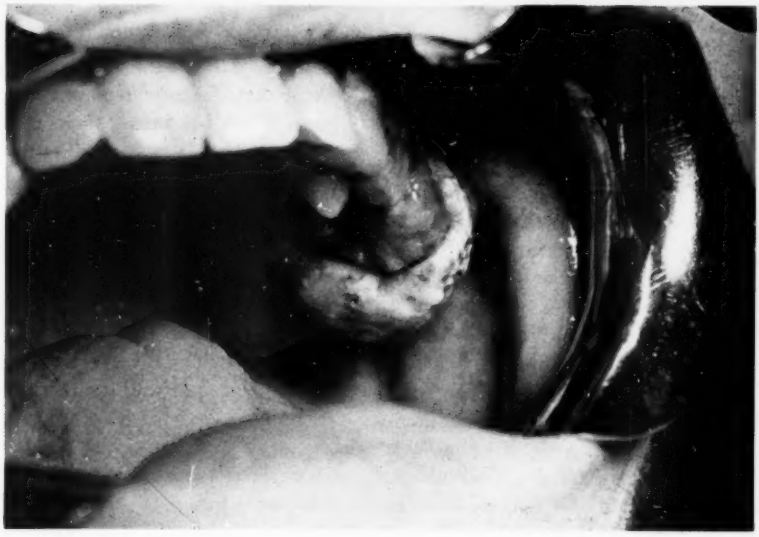

Case 3

Case 2

of the lesion and cauterization of the base. Six months after excision the patient became pregnant and delivered a normal baby at term. The lesion has not recurred during the past five years.

Case 2. This 48 year old male presented with a pedunculated growth on the buccal gingiva opposite the lower left first and second bicuspids. The lesion had been present for five weeks. Oral hygiene was poor and the radiograph of this area revealed severe alveolar bone loss around the teeth in this quadrant. The lesion was completely excised and the base was cauterized. Microscopic study revealed pyogenic granuloma with some new bone formation within the lesion. The growth has not recurred in three and one-half years.

Case 3. A 53 year old female had noticed a vascular, soft tissue growth and an associated hard calciferous lump in the upper left quadrant of her mouth for four or five months. Both masses gradually had increased in size. She was frightened and concerned about malignancy. The upper left second molar was missing and the first and third molars were entirely covered with a mass of salivary calculus. The lesion arose between the second bicuspid and first molar and nearly covered the carious crown of the second bicuspid. Radiographically no bone invasion was apparent, but periapical and periodontal disease were present. The lesion was excised and the posterior teeth in that quadrant removed for other reasons. There has been no recurrence in the three and one-half years since removal.

Case 4. This 14 year old female was afflicted at birth with Sturge-Weber's syndrome.[2] There was a large right facial hemangioma which also involved the mucous membranes of the right side of the mouth. The patient suffered seizures due to a large hemangioma of the right side of the brain, and she had a severe hemiplegia of the left side of her body. The growth from her right alveolar ridge had been removed several times and treated with radon once, but each time it recurred. Finally, extraction of the teeth in this quadrant and complete excision of the mass, including the adjacent hemangiomatous mucosa, was successful. There has been no recurrence for more than three years. In this case, it seems that the hemangiomatous condition of the gingivae predisposed to the development of the pyogenic granuloma.

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Bruxism, Diagnosis and Treatment

by DONALD S. MOORE, D.D.S.,* *Hamilton, Ontario*

BRUXISM is the term used to describe clenching or grinding of the teeth, either during the day or at night. It usually is a subconscious habit of which the patient is unaware. Bruxism is one of the most important etiological factors in the production of periodontal disease and in the loss of teeth. From a study of 606 cases the apparent causes of bruxism are as follows:

1. Tension.
2. Habit.
3. Hyperthyroidism.
4. Worry and hurry.
5. Interference in the occlusion.
6. Gastric and intestinal disturbances.
7. Anger.—The Old Testament refers to "weeping, wailing and gnashing of teeth."
8. Occupational.
9. Clenching, in order to keep the mouth closed to overcome snoring or mouth breathing.
10. Inflamed periodontal membranes, which create the urge to clamp down on the teeth.
11. Children with nasal and pharyngeal infections.
12. Heavy metal palates of partial and full upper dentures give the wearer a sensation that the denture is dropping at the distal, so that the patient clenches to overcome this sensation.

Over fifty per cent of the author's patients who have periodontal disease clench or grind their teeth either during the day or night, or both day and night. Robert L. Parrish, head of the Department of Periodontia at the University of Tennessee also stated (personal communication) that the prevalence of the habit is extremely high amongst the patients he sees.

Ninety per cent of the patients who clench their teeth are not aware of doing so. Because most patients are not aware of clenching or grinding their teeth, it is necessary for the dentist to be constantly on the lookout for bruxism. The following signs and symptoms should make one suspect the presence of the habit:

1. Patients with abnormally well-developed muscles of mastication should always be suspected of bruxism. However, if they are Slavic or of other European origin and eat foods that require considerable heavy mastication, this would not necessarily apply. (Fig. 2).

2. McCall's festoons at the gingival margin of the teeth.

3. Worn facets on areas of the teeth that do not touch during normal chewing excursions. This can be ascertained by giving the patient some wax to chew and noting whether he uses short or wide lateral excursions.

4. The luxation of the teeth bucco-lingually under grinding pressure.

5. Rigid type jaw muscles, which make it difficult to retract the cheeks.

6. Recessions.

7. Sensitive root surfaces.

8. Temporomandibular joint disturbances.

9. Loss of the alveolar bone around teeth. Bone loss around the second molars should always make one suspicious of either premature contact from an adopted centric, interference on the balancing bite or more commonly, clenching of the teeth. The fulcrum point is approximately two inches distal from the lower incisors, which means that the greatest pressures can be exerted by the muscles of mastication in the regions of the second molars. This is in accordance with the laws of leverage.

10. The contraction of the masseter or temporalis muscles observed while taking the case history. Also, the case history often will suggest the possible presence of bruxism, if gastric or duodenal ulcers, colitis, bad nerves, difficulty in sleeping, gagging readily, or other conditions that have tension as an underlying etiological factor are present.

*Associate in Dentistry, Department of Periodontology, University of Toronto. Presented at Chattanooga, Tennessee at the Thirteenth Annual Seminar of the Tennessee State Dental Association, July 1955.

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11. Teeth that are hyper-sensitive to vitality tests where there are no cavities or fillings present.

12. Pain within the alveolar process.

13. Blanching of the gingival tissues when the teeth are under biting stresses.

The next step is to ask the patient to note whether they wake up with their teeth together, or notice them clenched during the day. The patient usually will say he has not noticed the habit and he is quite sure that he does not do it. It is advisable never to tell the patient he has such a habit, but rather to say, there are symptoms in the mouth that suggest bruxism is present. If the dentist insists that patients clench or grind their teeth, some will come back and claim they have just acquired the habit as a result of the dentist's suggestion, and blame him for starting something that previously was not present.

Bruxism may be initiated by the dentist if a filling is left high in centric and the patient tries to bite it into place. The patients sometimes state that when they mention a filling being high the dentists have suggested that they bite down on it until it is comfortable. Partial dentures which do not quite occlude in centric will create the desire to get the teeth to meet and the habit may be initiated. Rests on partial dentures that are high in centric or lateral excursions can also produce bruxism.

BRUXISM IN CHILDREN

The habit of bruxism during childhood is not ordinarily a destructive process. The alveolar bone during childhood is more adaptive and absorbs the trauma more readily. The child's maintenance and reparative processes are far superior to those of an adult. When bone destruction does occur in childhood, it usually affects the permanent incisors and first molars since these are the first permanent teeth to erupt (precocious advanced alveolar atrophy); the child usually has "St. Vitus dance" (chorea) or some other nervous disease (Fig. 1).

Fig. 1. 18 year old female. Chorea. Clenching. No other systemic factors present. Note bone destruction.

The damage that results from bruxism for any mouth depends on how much force can be applied by the muscles of mastication and the frequency and duration with which this force is applied. If the teeth wear from grinding so that the interference in the occlusion is eliminated, the alveolar bone destruction will be slight or absent (Fig. 2). If the teeth drift away from the forces applied, there will also be little bone destruction (Box). This does not refer to the drifting of teeth after the alveolar bone has been destroyed and there is not sufficient bone to hold the teeth in the line of the arch.

When the maintenance and reparative processes of the body become lessened due to age, disease, or when there are other debilitating factors operating in the mouth, bruxism, which was not severe enough to cause bone destruction by itself, will then induce the loss of the supporting bone. With some individuals, slight clenching or grinding will actually produce denser, more heavily calcified bone, while with other

Fig. 2. Bruxism. Cheek biting. Well worn teeth. No bone destruction except around 2nd molars. Patient lived in Greece until 18 years old. Well developed arches and muscles of mastication.

patients, clenching is so severe that the observant dentist can actually see the tissues blanch when the teeth are clenched. Greater destruction of course, will occur where the stresses are being applied obliquely than where they are being applied along the long axis of the tooth (axial). The more the muscles of mastication are used, the stronger they become, which is in accordance with Wolfe's law, "Function determines form." Thus, the longer the habit persists, the greater will be the force that can be applied against steadily weakening supporting structures. The extra alveolar arm, that is, the part of the tooth above the edge of the surrounding alveolar bone, will become longer at the expense of the intra-alveolar arm, that is, the part of the tooth supported by bone. Thus the fulcrum point will move apically until the torque will cause even greater bone destruction and marked mobility of the teeth.

All tissues in the body require rest. Clenching overloads and overtires the supporting structures of the teeth and cuts off the circulation. When the forces exerted by the muscles of mastication exceed the physiological limits of tissue tolerance, tissue destruction results. It is most important for the dentist to bear in mind that bruxism is not confined to the natural dentition. The flattening of alveolar ridges beneath dentures is a very common, but rarely diagnosed result of bruxism.

Clenching of the teeth is a common occupational habit, particularly for operators of tractors, cranes, bulldozers, etc. It is also common for persons lifting heavy objects; e.g., shipping clerks. Any physical exertion induces some people to tense every muscle in their body, including the muscles of mastication, which are the most expressive muscles in the body. Aggressive businessmen and office workers are as prone to clench their teeth as they are susceptible to develop a nervous stomach or stomach ulcers. Dentists are particularly apt to clench their teeth because of the tensions produced as a result of practicing dentistry.

Mothers with young children are especially susceptible, as are single women in their late thirties, with marital intent. During the menopause, clenching is prevalent. The perfectionist or overly-conscientious female usually has the habit. These same individuals complain of tension headaches and tense muscles in the neck.

TREATMENT

The treatment of bruxism in the past has not been easy. The correct diet, rich in calcium, phosphorus and Vitamin B and D is important. To quote Dr. Tom Spies, "For many years psychotic changes have been associated with severe pellagra, but we have

learned that subclinical and mild pellagra frequently present symptoms which are diagnosed as hysteria, neurasthenia or anxiety states. The study of thousands of cases has shown that these symptoms often have certain characteristics which are useful in an early diagnosis of deficiency disease. They disappear dramatically following specific vitamin therapy only to return eventually if the patient remains on his usual inadequate diet without a supplement."[9] Dr. Spies also wrote: "The most frequent emotional outbursts are concerned with fear, apprehension, anger, hostility, depression, extreme sensitivity, and general emotional instability. Sometimes these persons appear to have what physicians might call 'simple anxiety states,' or the picture may be that of 'anxiety neurosis,' or anxiety hysteria. Nearly always such persons have insight, and in discussing what we call their pseudo hallucinations they often state, 'It was just my imagination.' Almost without exception, patients complain of a poor memory. Saline injections produce no benefit but administration of thiamine and nicotinic acid result in truly amazing personality changes. Within thirty minutes to twenty hours, a timid, apprehensive, shrinking person becomes a smiling and co-operative individual."[9]

Gastro-intestinal disturbances must be overcome with the cooperation of the physician. It is not unusual for adults to have intestinal parasites and their elimination is necessary for successful therapy.

Many patients stop the habit when the occlusion is equilibrated, or when the partial dentures are relined and the interference of the rests removed. If metal is to be used in the palate of a full upper denture or in a partial upper denture with free end saddles, the metal should be very thin to decrease the weight of the denture in order to overcome the sensation of the denture dropping. Some dentists prefer a palate of metal mesh and plastic to reduce this weight.

Tension is the commonest cause of bruxism and this is the most difficult type to overcome, because it requires the cooperation of the patient. All tension is expressed as muscle tension whether it is the result of worry, hurry, fear, frustration, or any of the emotions, and as mentioned previously, the jaw muscles are very expressive and are quickly affected by our emotions. A discussion with patients as to the cause of their tension is of paramount importance. It is necessary to find out why they are building up tension, in order to help them overcome the causes. Not all patients are desirous of help or willing to recognize that their trouble has an emotional background. Also some patients who will accept the fact that their trouble is emotional, will not co-operate in eliminating the cause. Some patients require the aid of a psychiatrist because of guilt complexes or other involved psychological disturbances. It is unfortunate that many of the patients who require psychiatric help are the most likely ones to object to being examined by a psychiatrist. A psychiatrist, who is a highly trained physician, should be seen before mental breakdown occurs, but the misguided public is under the impression that they should only be consulted after a mental breakdown. Psychiatry is a more fearful word to most people than dentistry. From experience it has been found that it is more diplomatic to suggest they seek psycho-therapy from a physician.

Fig. 3. Soft rubber or plastic appliance.

Fortunately, most patients are building up tensions due to less complex reasons

than those that require a psychiatrist's help. By convincing them to apply logic to their everyday problems of living they can learn to overcome tensions. With some patients, it is a case of discussing their fears and anxiety with a minister trained in personal counselling. In many cases, after the patients have had a chance to discuss their problem with the dentist, they find they feel much better and their tensions disappear.

The work of Dr. Hans Selye, of Montreal, on stress, has focused the eyes of the physician and dentist upon the effect of such stresses in producing disease. Since his "stress adaption syndrome" was published in 1947, there have been 19,000 articles on stress published in the medical journals. For years many physicians have been suspicious of the effect of stress on the body and Dr. Selye's experiments proved their suspicions to be true.

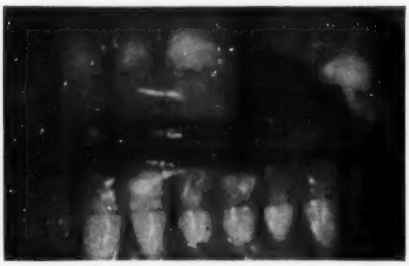

Fig. 4. Upper and lower splint.

Breaking the habit of bruxism consists of three phases.

1. A sympathetic understanding of the patient's problem.

2. Teaching the patient how to relax as outlined in various excellent books on relaxation.[1,2,3,4]

3. Providing the patient with a program for overcoming the tension as outlined in such excellent books as "The Power of Positive Thinking,"[5,6] by Dr. Norman Vincent Peale (the best seller of 1952). "How to Stop Worrying and Start Living,"[8] by Dale Carnegie, and many other excellent books.

Since the habit is subconscious, one can use the patient's subconscious mind in helping to overcome it. This can be accomplished by autosuggestion, using the same principle that patients would use if they desired to get up at a certain time in the morning when they did not have an alarm clock or anyone to awaken them. By concentrating on, "I must get up at a certain hour," and planting that idea in their subconscious mind, they would awaken at the desired time because the subconscious mind does not sleep and keeps track of time. The same principle can be used to control bruxism at night, whereby the patients concentrate as they fall asleep, "I must keep my lips together, but teeth apart." For this to be successful, patients must be given an incentive to overcome the habit, otherwise they would not concentrate well enough or long enough to plant the idea in their subconscious mind. Also for autosuggestion to be effective, the patients should be in a relaxed state, as then the subconscious mind is more receptive to suggestion. Therefore, there are two reasons why the patients must learn a technique of complete muscle and mental relaxation. First, so that they sleep in a relaxed state, and secondly, so that the autosuggestion will be effective. Most people are under the impression that when they sleep they are completely relaxed, but if such were the case they would not clench or grind their teeth, nor would they wake up with their hands clenched or their shoulder, chest, or leg muscles cramped.

For those patients who are not successful in breaking the habit it is necessary to make an appliance for them to wear at night, in order to reduce the effects of the habit. Different types of appliances are used depending on each individual case. If it is a case of clenching, a soft rubber or plastic bite positioner (Fig. 3) is made for the lower jaw. This acts as a cushion and prevents excessive pressures on the teeth and the resultant interference in the

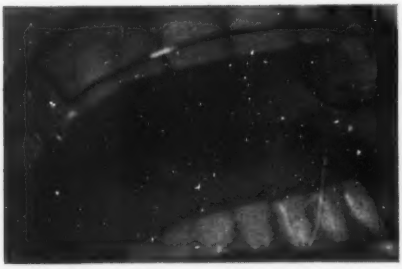

Fig. 5. Hawley type of appliance.

Fig. 7. Open bite—For posteriors in centric, created by bite opener worn to take the load off the centrals which were loose. Incorrect appliance and clenching depressed the teeth.

circulation to the supporting tissues. Any appliance made must not impinge on the soft tissues and because of caries it should not cover the gingival margins. Any periodontal prosthesis, if possible, should not encroach beyond the free way space. For some mouths it is better to construct clear acrylic bite planes for upper and lower jaw (Fig. 4) that will splint the teeth, prevent the luxation of the teeth, and provide a smooth flat surface.

An appliance similar to a Hawley bite plane is useful for those patients with deep overbites (Fig. 5). This allows the lower incisors to slide around on a flat plane. It has the disadvantage that sometimes where the bite is not closed, the posteriors overerupt leaving an open bite in the anterior region. Instead of using a labial wire as in Figure 5, it is usually better to run the acrylic up onto the labial surface as in Figure 6, and to cover the occlusal surfaces of the posteriors to prevent their overeruption.

The appliances used to reduce or eliminate the effects of bruxism must be worn as long as the habit persists. Since the appliances usually do not break the habit of bruxism, it is apparent that they have to be worn every night. It can readily be seen that there is a distinct advantage in motivating the patient to eliminate the causes and the habit of bruxism, rather than resorting to appliances. Also, if the patient clenches or grinds his or her teeth during the day, it is unlikely that it will be convenient to wear an appliance at that time.

It is quite amazing how quickly the patients get used to wearing an appliance.

Fig. 6. In place of Hawley appliance.

Fig. 8. Appliance in place. Posterior teeth depressed by clenching and by improperly designed appliance.

Fig. 9. An appliance made to take the load off the loose centrals but it impinged on the free way space. It did not help to alleviate the bruxism; in fact it made it worse as well as depressing the posterior teeth.

Most patients get so used to them that, if the appliance is misplaced, they feel lost without it. The Hawley bite plane type of appliance can be utilized to move anterior teeth into the position desired at the same time it is worn for bruxism. Regardless of what type of appliance is made, it is necessary that the occlusion of the appliance be checked most carefully (Fig. 7, 8, 9).

CONCLUSIONS

Since successful periodontal therapy depends on recognizing the causes for periodontal diseases and removing them, it is therefore necessary to diagnose and treat bruxism. In so doing, the local factors, as well as the systemic and psychological factors, must be considered. Due to the fact that bruxism may produce painful symptoms, the relief of such symptoms is of paramount importance to the patient and the dentist.

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SPRING CONFERENCE OF THE

AMERICAN ACADEMY OF PERIODONTOLOGY

The annual Spring Conference of the Academy of Periodontology will be held at the University of Michigan, Ann Arbor, Michigan, on April 29, 30, May 1, 1957. The program will be announced later.

Effect of Zinc Phosphate Cement Fillings on Gingival Tissues*

by JENS WAERHAUG, D.D.S., PH.D., *Oslo, Norway*

THE reaction of gingival tissues to subgingival zinc phosphate cement fillings has not been extensively studied. Accidental findings in autopsy material[1] seem to indicate that the epithelium under favorable conditions can join such fillings and that a membrane of an appearance similar to that of the secondary cuticle can be formed between the cement and the epithelium. In an experimental investigation on "The tissue reaction around artificial crowns"[2] fastened with cement, it was observed that acrylic crowns were well tolerated by the soft tissue, but that the cement apparently caused a more or less severe irritation. It was furthermore pointed out that an absolutely tight marginal sealing was difficult to obtain.

The present study is a link in a series of experiments set up with the purpose of evaluating the effect on gingival tissues of the different materials used in dentistry.

MATERIAL AND METHOD

Ten teeth in 3 monkeys, and one tooth in a dog were used for the experiment. All the animals were young and healthy and the gingivae were normal. For the sake of survey of the field of operation, the cavities were prepared on the buccal surface. The preparation was extended to the bottom of the clinical pocket.[1] During this procedure the soft tissue was more or less lacerated and to stop the bleeding the cavity was packed with cotton soaked in adrenalin. A steel matrix was contoured to fit the tooth surface in the area of the cavity, and this matrix was kept in place with finger pressure during the filling with cement. Immediately prior to the insertion of the cement,† the cavity was rinsed with a jet of water, washed with chloroform and dried with warm air. The fillings usually were polished one to two hours after the insertion. They remained in place for observation periods varying between 82 and 338 days. The animals were decapitated under nembutal anesthesia and the jaws were taken out. During this procedure care was taken not to move the gingivae in relation to the tooth and the fillings. The jaws were fixed in 10% formalin and decalcified in 5% nitric acid. After decalcification the jaws were cut to pieces of proper size. These specimens were embedded in celloidin and every tenth section was stained with hematoxylin-eosin.

OBSERVATIONS

The gingivae were examined regularly during the observation period. By visual inspection the outside of the gingivae looked normal within one week. However, in most cases some liquid could be pressed out of the pocket adjacent to the filling.

The six illustrations demonstrate the most typical conditions. In all cases the cement had been nearly completely dissolved during the decalcification (Figs. 1-6). In most cases the general contour of the fillings corresponded to that of the enamel surface (Figs. 2-5), but occasionally overhangs (Fig. 1) and shortnesses (Fig. 6) could be seen. A microscopic sealing of the cavity to the margin was not observed in any case in this series. The width of the crevice between the cement and the tooth could be as small as 10 micron (Fig. 1, right). As a rule the crevice between the preparation and the

*From the Institute of Dental Research, Josefinegate 32, Oslo, Norway. The experiments on which this article is based, were undertaken at the University of Minnesota, School of Dentistry. The author desires to express his thanks to Dr. W. Crawford, dean of the Dental School and Dr. H. A. Zander, chairman of the Division of Periodontology.

†The cement was chosen at random and happened to be Flecks Zinc Cement.

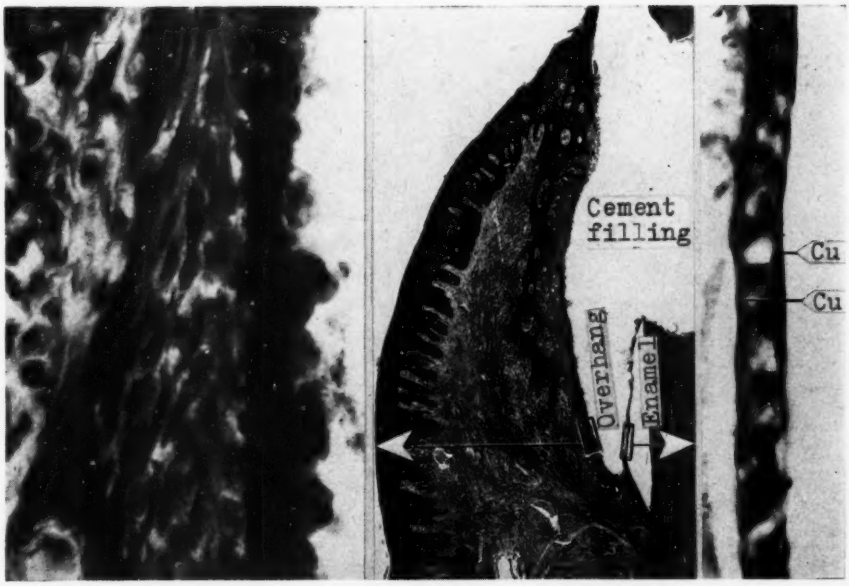

Fig. 1. M6, UL3. Observation period 319 days. Cement pressed over margin of preparation to form overhang. Between overhang and enamel 10 micron wide crevice filled with polymorphonuclear leukocytes and bordered on both sides with cuticles, Cu., (right). Approximately normal epithelial cells bordering upon keratinous membrane on surface of cement overhang (left).

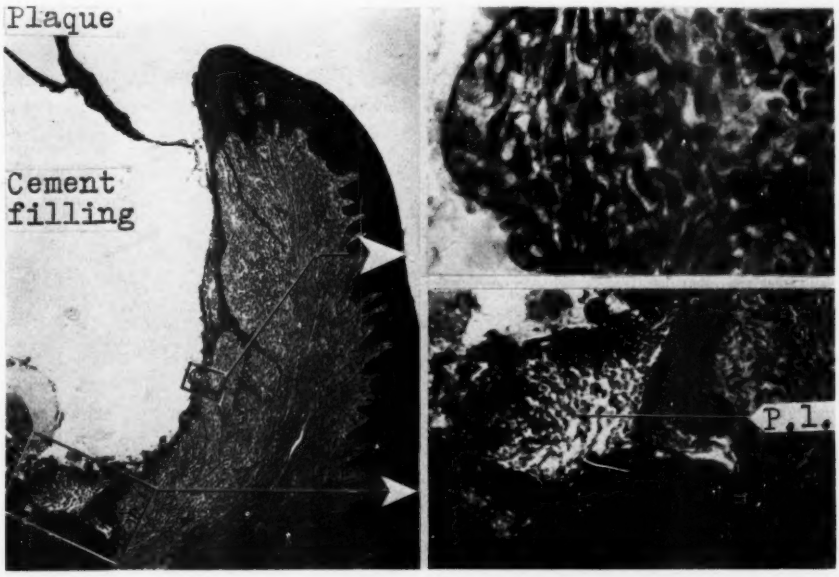

Fig. 2. M6, LR5. Observation period 160 days. Crevice between cement and preparation filled with debris and polymorphonuclear leukocytes, P.l., (bottom right). No downgrowth of epithelium below margin of cavity. Epithelial cells showing degeneration border upon "cuticle" on surface of cement (top right).

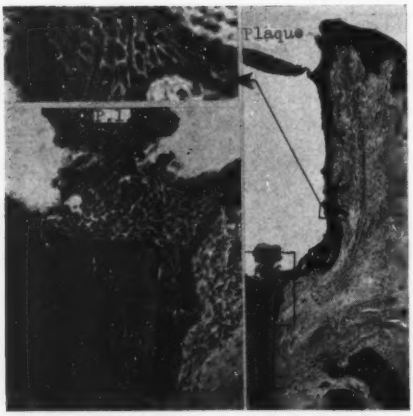

Fig. 3. M5, LR5. Observation period 216 days. Crevice between cement filling and preparation filled with debris, epithelial cells and polymorphonuclear leukocytes (bottom left). Downgrowth of epithelium below cavity margin (0.4 mm.). Slight inflammation in connective tissue, ending at deepest point of epithelium. Approximately normal epithelial cells bordering upon keratinous membrane on surface of cement (top left).

filling material was widest near the margin and decreasing inward (Figs. 2-6). In the crevices there could be found debris and polymorphonuclear leukocytes (Figs. 1-4) and/or bacterial plaque (Figs. 5 and 6). In one case (Fig. 4) blood or tissue fluid may have flowed into the cavity during the insertion of the cement, as an organic framework (X) is here seen in the cement. Outside this there is an accumulation of bacterial plaque (right).

In cases where no plaque was present on the cement filling, a membrane, apparently of epithelial origin, was found between this and the epithelium (Figs. 1, left, 2, top right and 3, top left). The thickness of this membrane varied considerably and often crystals of the cement were embedded. When such membranes were present, epithelial cells usually were found bordering directly upon them. These epithelial cells could sometimes look very close to normal (Figs. 1, left and 3, top left), although a certain amount of degeneration was common (Fig. 2, top right). In the connective tissue under such areas there was some infiltration of lymphocytes and plasma cells.

When bacterial plaque was found on the fillings, epithelial cells never were found bordering directly upon this; there were always polymorphonuclear leukocytes in between (Figs. 4-6). The degeneration of the epithelial cells and the inflammatory response in the connective tissue were conspicuously more pronounced in areas where plaque was present than in areas where the soft tissue bordered directly upon the cement.

In Table 1 a few data have been summarized. It may be seen that the average downgrowth of epithelium below the deepest point of the preparation was 0.35 mm. The most extensive downgrowth was observed where bacterial plaque was present (Figs. 5 and 6). In the cases where no plaque was present, no downgrowth had occurred (Fig. 2). The filling was completely covered by bacterial plaque in 5 cases and partially in 3 cases. In 3 cases no plaque was present. Plaque was found between the filling and the preparation in 7 cases.

DISCUSSION

This series corroborates earlier findings[2] that a microscopic sealing of the cavity is difficult to obtain. The crevices between

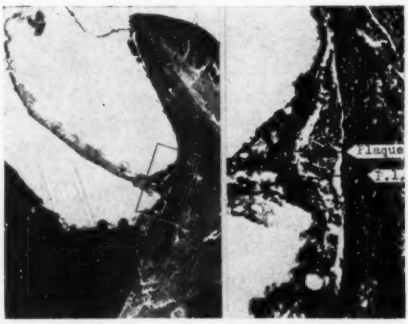

Fig. 4. M6, LL5. Observation period 187 days. During insertion of cement, blood or tissue fluid may have flowed into cavity, leaving organic matrix (x) in cavity after dissolution of cement. Corresponding to organic matrix is formed plaque on surface of cement (right). Between plaque and degenerated epithelium are polymorphonuclear leukocytes, P.l. Severe infiltration of lymphocytes and plasma cells in connective tissue corresponding to plaque.

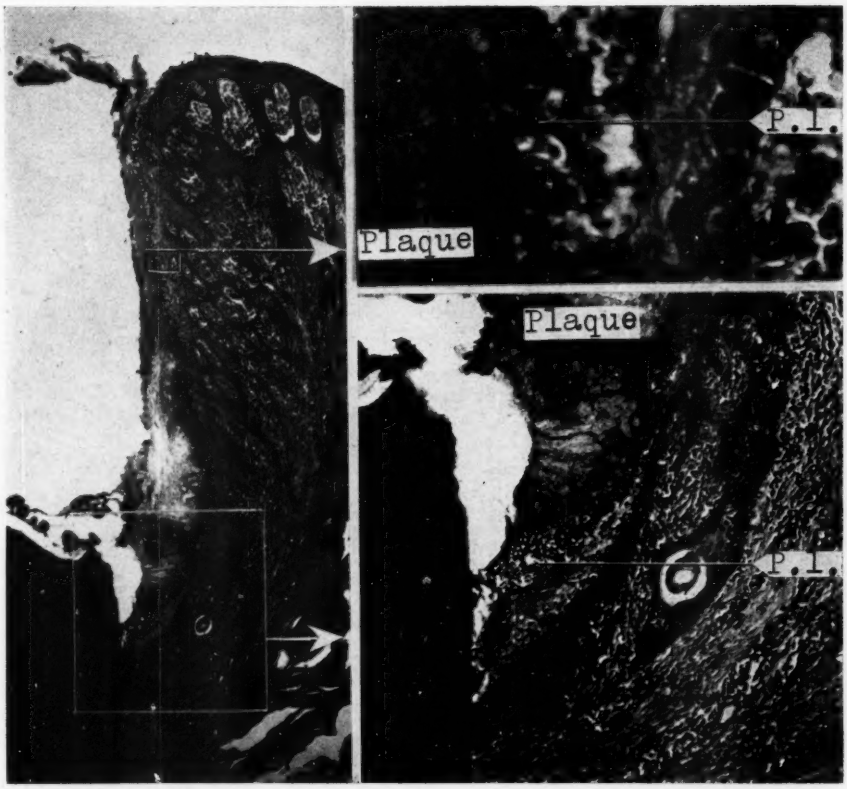

Fig. 5. D13, LR4. Observation period 82 days. Crevice between cement and preparation and entire surface of cement filling covered with bacterial plaque. Between plaque and degenerated epithelium are polymorphonuclear leukocytes (right). Severe infiltration of lymphocytes and plasma cells in connective tissue extending below deepest point of epithelium.

the tooth and the cement obviously represent weak points. Cells may degenerate and die in them and the waste products will irritate the soft tissue (Figs. 2 and 3). The inflammation thus produced is mild when compared with that produced by bacterial plaque, and under favorable conditions a destruction of periodontal fibers with ensuing downgrowth of epithelium may not occur (Fig. 2). However, the most important danger combined with these crevices is that bacteria are retained in such a way that they cannot be attacked by the polymorphonuclear leukocytes. Therefore, a bacterial plaque is allowed to be formed (Figs. 4-6). This plaque represents a much more potent irritant than the cement and/or the products of degeneration, and besides that a bacterial plaque has the ability to migrate apically (Fig. 6). It is interesting to note that the amount of downgrowth of epithelium below the deepest point of the preparation was rather constant (0.4 – 0.5 mm.) in the cases where the cement filling was covered by plaque. There seems to be little doubt that this downgrowth is caused by the inflammation engendered by the bacteria (Figs. 5 and 6). Even the slight inflammation caused by the cement alone is sufficient to break down the periodontal fibers and allow downgrowth of epithelium (Fig. 3).

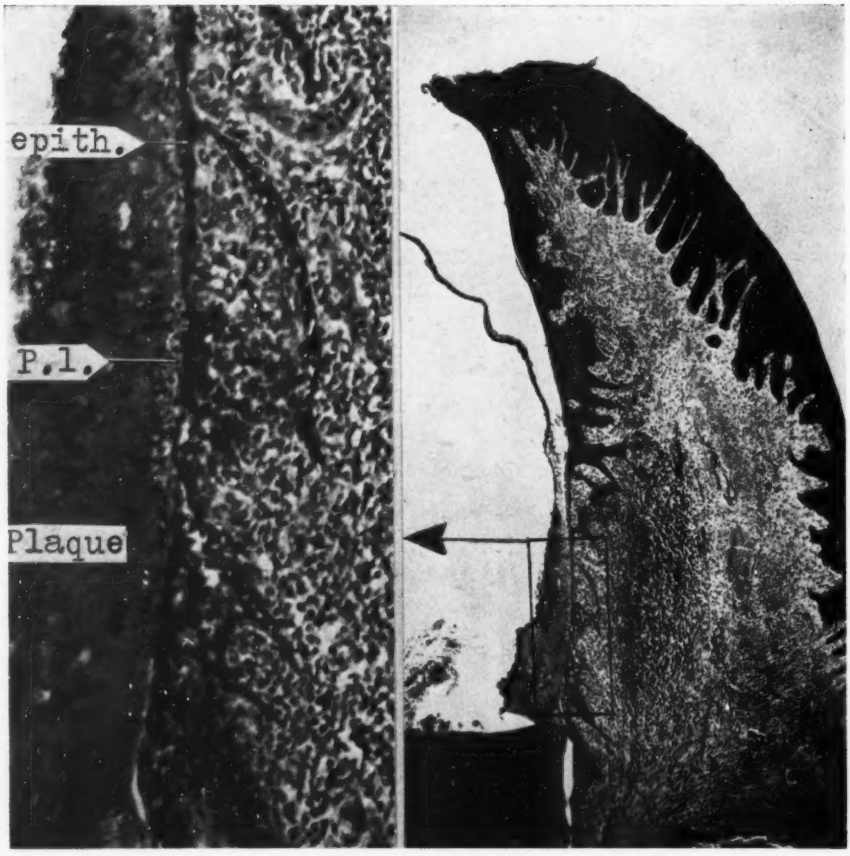

Fig. 6. M5, UL5. Observation period 216 days. Surface of cement filling covered by bacterial plaque. Between plaque and degenerated epithelium are polymorphonuclear leukocytes (left). Severe inflammation in connective tissue.

Even in those cases where no plaque was present there was some degeneration of the epithelial cells bordering upon the cement and some inflammation in the connective tissue. How much of this irritation is caused by the cement itself, and how much by the products of cell degeneration is not possible to decide. It is not likely that the cement acts as a mechanical irritant as the best adaptation of the soft tissue to the cement was found on an overhang (Fig. 1). This is in accordance with earlier findings concerning "the effect of rough surfaces upon gingival tissues."[3] Therefore, the irritation, if any, seems to be mainly chemical. However, zinc cement cannot be a strong irritant as more or less normal epithelial cells in that case would not be able to adjust themselves to it like an epithelial cuff (epithelial attachment). It is possible that the thick deposition of a keratinous substance similar to the "secondary" cuticle may act as isolation (Figs. 1 and 3) against the chemical irritants.

This experiment seems to throw more light upon the formation of the so-called "secondary cuticle": The irritation exerted

TABLE 1

Data concerning observation period, downgrowth of epithelium below preparation, presence of bacterial plaque on surface of filling and between filling and preparation in 11 teeth in one dog and 3 monkeys, where zinc phosphate cement fillings had been placed.

Animal	*Tooth*	*Observ. period*	*Downgr. of epithelium below prep. in mm.*	*Presence of plaque on filling*	*Plaque between filling and preparation*
D 13	LR 4	82	0.4	+	+
M 4	UL 5	338	0.5	+	+
M 4	LR 5	225	0.4	+−	+−
M 4	LL 5	154	0.4	+	+
M 5	UR 5	216	0.5	+	+
M 5	UL 5	216	0.0	−	+−
M 5	LR 5	216	0.4	+−	−
M 6	UR 5	160	0.5	+	+
M 6	UL 3	319	0.3	−	−
M 6	LR 5	160	0.0	−	−
M 6	LL 5	187	0.4 (0.0)	+−	−

by the cement may cause a degeneration of the superficial layers of the epithelium and the degeneration products may then be deposited on the surface of the cement as a membrane of keratinous nature similar to the "secondary cuticle." This finding seems to corroborate the supposition that the "secondary cuticle" is built up from the degenerating superficial cells of the epithelial cuff.[1,2,4] As far as the formation of a keratinous membrane and the adaption of the epithelium to this are concerned, the reaction seems to be similar in dog and man.[1] There is also reason to believe that the formation of bacterial plaque and the reaction to this are governed by the same rules in man, dog and monkey.

When a comparison is made between heat-cured and self-cured acrylic resin and zinc cement, it is obvious that the zinc cement is more irritating than the heat-cured resin,[2,5] but less irritating than the self-cured resin.[6]

CONCLUSIONS

1. Zinc phosphate cement may under favorable conditions be rather well tolerated by the epithelium although some irritation always is present.

2. The irritation seems to be chemical in nature and not mechanical.

3. Zinc phosphate cement seems to be more irritating than heat-cured acrylic resin, but less irritating than self-cured acrylic resin.

4. The epithelial lining of pockets around zinc cement fillings may adhere directly to a membrane on its surface like an epithelial cuff (epithelial attachment). However, the superficial layers of the epithelium show some more degeneration.

5. It seems to be difficult to make a deep subgingival cement filling absolutely tight at the margins.

6. Crevices between the filling and the preparation may be filled with debris and degenerating cells, and the degeneration products may cause a comparatively slight inflammation in the adjacent soft tissue.

7. The crevices may harbour bacteria

in such a way that they cannot be attacked by the polymorphonuclear leukocytes, thus allowing a bacterial plaque to be formed on the filling.

8. Overhangs do not seem to act as mechanical irritants.

9. A keratinous membrane similar to the "secondary cuticle" can be formed on the surface of cement fillings.

SUMMARY

In 11 teeth in 3 monkeys and 1 dog, zinc phosphate cement fillings were inserted in cavities prepared to the bottom of the clinical pocket. After 82 to 338 days, it was observed that under favorable conditions an approximately normal epithelial cuff could be formed on the cement in limited areas. Zinc cement seems to exert a slight irritation mainly of chemical nature. Crevices of different width were always found between the filling and the tooth, and in these crevices cells degenerated, or bacterial plaque was formed causing more or less severe inflammation in the adjacent tissue. In most cases the entire cement filling was covered by a continuous mat of bacteria and the inflammatory reaction was completely governed by these.

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A Method of Producing Artificial Calculus on Typodont Teeth

by DONALD E. KNAPP, B.S. PHARM.,* *Ann Arbor, Michigan*

THE teaching of scaling technic to dental students is frequently accomplished with the aid of a typodont having Ivorene teeth and soft rubber gingiva. This method is very satisfactory for teaching instrument and hand position. The method is much more realistic and the accomplishment greater if artificial calculus can be placed on the teeth in a supra and subgingival position. By this method the student can be taught tactical skills which are so important in scaling.

There have been a number of attempts in the past to create an artificial calculus which could be applied to typodont teeth and would exhibit the physical properties of the natural substance.

As an initial list of requirements for an artificial calculus, it was decided that it should possess:

1. Good adhesion
2. Hardness
3. Some fracture tendency
4. A fairly rapid set
5. A grainy feeling under the scaler
6. No surface gloss on drying
7. Ease of application
8. Low cost

More or less empirically adhesives of various kinds were tested to determine what materials would adhere to the Ivorene typodont teeth. Many kinds of varnishes, shel-

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lacs, lacquers, cements, resins, glues, gums (synthetic and natural), and glazes were tested, but none of those tested possessed sufficient adhesion when modified to obtain the desirable physical properties.

From the Columbia Dentoform Corporation it was learned that the Ivorene plastic was a urea-formaldehyde resin. U.F. resins have widespread application in home and industry, but probably the best known is formica. The logical course then was to determine what was used to glue formica. There are two materials in common use, one a contact bond cement and the other known commercially as urea resin glue.* Contact bond cement dries to a sticky gum and could not be altered to yield hardness. Urea resin glue when mixed with water and allowed to set hardens very nearly as hard as the Ivorene tooth. This necessitated rather specific requirements for a diluting medium which would soften the mass yet not greatly inhibit the bond. Therefore it must:

1. Be insoluble but wettable in water
2. Have fairly uniform particle size
3. Be soft
4. Be compatible with urea resin glue
5. Be relatively non-hygroscopic

After some consideration it appeared that *powdered* asbestos would fulfill these requirements. A series of mixtures with resin/asbestos ratios of 1/0.5, 1/0.75, 1/1.0, 1/1.25, 1/1.50, 1/1.75 and 1/2.0 were mixed with sufficient water to yield a smooth paste and applied to typodont teeth. After twenty-four hours at room temperature there appeared to be no appreciable change in hardness. The resin/asbestos ratio of 1/1.75 appeared quite satisfactory but lacked grainy texture. Introducing pumice into the mixture and altering the resin/asbestos ratio finally produced a product which seemed ideal. The recommended mixture would contain:

Urea resin glue	1 part
Asbestos	1.5 parts
Pumice	.5 parts

After the powders are thoroughly mixed, they should be kept dry in a sealed container. In this manner the material can be kept indefinitely. When water is mixed with the powders in the proportion of 1.5 cc/level teaspoonful, a creamy paste results which may be easily applied to the typodont teeth. (Since oils or greases inhibit the bonding, prior cleansing of the typodont teeth with a little acetone or other organic solvent is recommended.) The teeth can be removed for the application of the material and may be replaced in the typodont two hours after application if desired. If the material is painted on the teeth while they are in the typodont, contact with any roughened areas on the rubber base should be avoided, or the rubber base may be coated with a thin film of cold cream, brushless shaving cream, or similar substance. A twenty-four hour period should be allowed for hardening at room temperature. If heat acceleration of the set is used, the period may be shortened to a half hour; but a harder end-product results and the resin/asbestos ratio must be altered depending on the temperature used. Too high a temperature will result in bubbling of the mixture and loss of desired physical properties.

*Urea-resin glue is a urea-formaldehyde resin powder which polymerizes after the addition of water. The polymerization proceeds fairly rapidly at room temperature and can be cold inhibited or heat accelerated.

I wish to thank Dr. D. A. Kerr for arousing my interest in calculus, Dr. J. K. Avery for suggesting the attempt to improve artificial calculus and Dr. D. G. Hard for her assistance in defining the desired physical properties in a calculus substitute.

The Relation of the Topography of the Root Surface to the Removal of Calculus

by SANFORD C. FRUMKER, D.D.S.,* AND WILLIAM M. GARDNER, D.D.S.,** *Cleveland, Ohio*

IN contrast to the many areas of disagreement in periodontology, there is one area significant because of the amount of agreement over it. This is the necessity in all cases for the complete removal of all calculus.

The relation of calculus to periodontal inflammation and destruction has been recognized from ancient times on. It is most rewarding and interesting to see the references to calculus throughout the history of dentistry.

Possibly the earliest reference in the literature dates way back to the middle of the 7th Century where Paul of Aegina writes that tartar incrustations must be removed either with scrapers or by small files.[9] Albucasis (936-1013) recommended a thorough cleansing with a set of 14 scalers devised by him.[12] Celsus in 1478 writes about the importance of scraping teeth clean.[6] The great Eustachius in 1563 listed tartar as a disease of the teeth.[12] Jacques Guillemeau in about 1600 gave a minute description of how to remove calculus,[2] and Fauchard's description of pyorrhea and its treatment, published in 1746, is today still a classic.[2]

In the United States the history of calculus has been even more fascinating. In 1768 John Baker, one of America's earliest and most famous dentists announced his arrival in New York and stated that "he cures scurvy of the gums . . . cleans and scales from the teeth that corrosive tartarous gritty substance." In 1784 "Dr. Fendall cures the most inveterate scurvy in the gums by careful scaling." John Greenwood, dentist to George Washington, in 1799 described how tartar "will insinuate itself between the gums and the teeth and separate them by degrees."[13]

However, it was not until the 1860's and the monumental work of John M. Riggs that periodontia took its greatest step forward. Riggs' work was so spectacular that he is rightly considered the founder of modern periodontia,[7] and for many years until the introduction of the term pyorrhea alveolarus,[10] periodontal disease was called Riggs' Disease. While not a prolific writer, the clinics given by Dr. Riggs, which stressed calculus removal, were very influential.

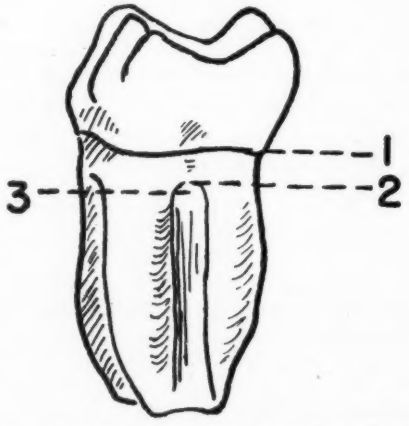

Fig. 1. Mesial aspect of the lower first molar. Line #1 is the height of the CE junction, line #2 the beginning of the furrow, and line #3 the depth of the furrow.

The scaling techniques of Riggs' were carried to near perfection by the renowned William J. Younger, and today virtually all the texts in periodontology stress the importance of calculus removal.[1,3,4,5,8] Very recently, the excellent experimental work of Waerhaug placed even more emphasis on calculus.[11]

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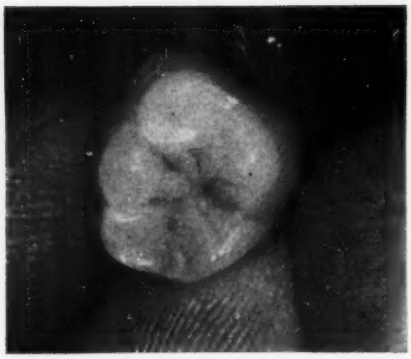

Fig. 2. A sickle scaler riding on the ridges of a furrow on the mesial surface of the root of a lower first molar. The sickle scaler gives a smooth feeling, but does not touch any calculus in the depth of the groove.

Many factors pertaining to the successful removal of calculus have been discussed at length. However, one factor of great importance has received, at least in the literature, but minimal mention. This important factor is the topography of the root surface from which we are attempting to dislodge the calculus.

OBSERVATIONS

The root topography to which we refer is the various grooves, dips, and curves in the root surface. As destruction results in a greater clinical exposure of the root, we run into more of these factors and an awareness of the root topography becomes increasingly important. Possibly the most common offender is the mesial root of the lower first molar, where there is usually a furrow along the long axis.

Figure 1 is a drawing of the mesial aspect of the lower first molars. All measurements are the average of 37 lower first molars measured by us. The crown length was 7.8 mm. and the root length 13.3 mm. The distance between line #1, the height of the CE junction, and line #2, the beginning of the furrow, is 2.8 mm. Line #3 represents the depth of the furrow. The distance between the beginning of the furrow (line #2) and the depth (line #3) is 0.5 mm.

Thus, between lines #1 and #2 we have an area easily accessible to a scaler, but by the time we reach line #3, only 3.3 mm. from the height of the CE junction, we are in a furrow from which the removal of calculus can be extremely difficult. The root in cross section at line #3 forms a dumb-bell shape. This becomes of great importance when we realize the instrument most commonly used to scale this surface is the straight interproximal sickle scaler. This instrument can easily ride on both margins of the furrow, give a smooth feeling, but leave the depth of the furrow filled with calculus. Figure 2 shows an instrument riding on either side of this furrow without being anywhere near its depth.

Another area that can cause difficulty is a groove that leads to a bifurcation. The worst offender is the upper first bicuspid. When it is bifurcated we usually find a

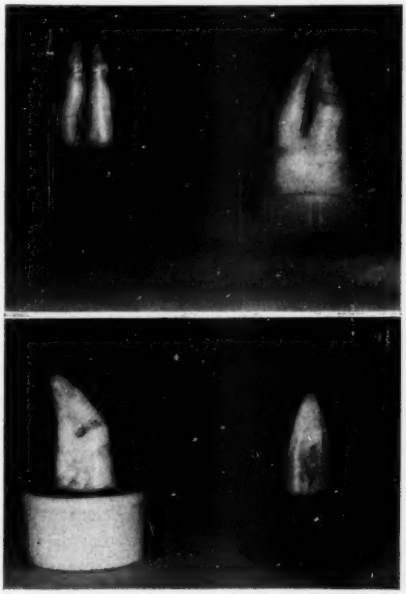

Fig. 3. Upper. On the left, a bifurcated upper first bicuspid with a deep, narrow groove leading into the bifurcation. To the right, a lower molar with the groove on the buccal. Lower. To the left, calculus in the crotch of a severe bend in the root of a cuspid. Note how well polished and planed the root is, but the calculus remained. To the right, a calculus filled furrow in a lower incisor.

deep and narrow groove, especially on the mesial, leading into the bifurcation. Figure 3 (upper) to the left shows such a bicuspid, and to its right is a lower molar exhibiting the groove on the buccal.

Figure 3 (lower) to the left, shows a cuspid with a severe bend in its root, and in the crotch of this bend rests a large piece of calculus. To its right is a lower incisor which has a furrow filled with calculus. Figure 4 to the left shows a lower molar with calculus remaining on the corner, the most rounded part, of the root. To its right is another grooved upper bicuspid.

Fig. 4. On the left, calculus on the corner of the root of a lower molar. To its right, a grooved upper first bicuspid.

DISCUSSION

All the teeth in the illustration were scaled prior to extraction. Note how shiny and well planed the root surfaces are, but note also how each piece of calculus is either in an inaccessible place, or in an area in which it would give a smooth feeling. The cuspid in Figure 3 (lower) is especially viscious as the calculus here gave exactly the feeling of a smooth bend in the root. Thus, should gingival inflammation continue around a tooth after treatment, it is important to investigate the topography of the root. Should there be any grooves or bends, they must be considered having calculus until proven otherwise.

The molar in Figure 4 shows the area most commonly missed in ordinary scaling, the corner and most rounded part of the root! It is here that our instruments are working most tangenitally to the surface, and it is here that we must be especially careful.

The mesial furrow on the lower first molar may present difficulties more serious than the much publicized bifurcation involvement. Many of these furrows are more difficult to maintain than many open bifurcations. The grooves and bends in roots can make the complete removal of calculus extremely difficult. When the calculus is removed there still remains an area that is particularly prone to calculus reformation, and is most difficult for the patient to keep clean. Such areas must be pointed out to the patient so he can pay particular attention to them during his home care.

The knowledge of the topography of the root is important in making a prognosis for an individual tooth. Should any of the illustrated difficulties be encountered, the dentist must pay special attention to these areas. The discovery of grooves can many times dictate the choice of treatment. Prepacking or gingivectomy with resultant scaling by vision are sometimes the only sure methods of cleaning out such grooves. In addition, the presence of grooves will necessitate a more often recall of the patient in order to be sure these areas are kept free of calculus.

Much can be learned from studying extracted teeth. Nothing is learned from a tooth immediately thrown into a waste basket. However, especially on teeth around which periodontal treatment has failed, if the tooth is observed the cause of the failure is often obvious. The cause will usually be calculus which has not been removed because it is hidden in any of the above mentioned variations in the topography of the root surface.

SUMMARY

1. Due to the topography of the root surface upon which we are working it may be very difficult to completely remove all the calculus present.

2. Furrowed roots, grooves, and dilacerated roots cause the most trouble. The rounded corners of the roots are also very often missed in scaling.

3. The topography of the root will effect the prognosis of the tooth, the choice of treatment plan, and the amount of allowable time between recalls of the patient.

4. Much can be learned from studying extracted teeth and noting where the calculus is lodged.

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DR. VIRGIL LOEB

Doctor Virgil Loeb, well known member of the Academy of Periodontology and diplomate of the Board of Periodontology, died on June 26, 1956 in St. Louis. Dr. Loeb practiced dentistry in St. Louis for almost fifty years, and taught both medical and dental schools there, since he had earned both medical and dental degrees.

Dr. Loeb found time to be active in organized dentistry, serving as president of the St. Louis Dental Society (1916) and the Missouri Dental Association (1935-36). He will also be remembered for his many contributions to the Academy of Periodontology.

Histological Results of Root Curettage of Human Teeth

by ERWIN M. SCHAFFER, D.D.S., M.S.D.,* *Minneapolis, Minnesota*

IN the literature, there are numerous reports on the histological effects of subgingival curettage of the soft tissue wall of periodontal pockets.[1,2,3] Conversely, very little work has been done on the effects of curettage of the root surfaces.[4,5]

The following are some of the most debated questions concerning root curettage of teeth. Should any cementum be removed in order to have a root free of calculus? If the cementum must be removed, how much removal is necessary? Is it possible to remove one-third or any other predetermined fraction of cementum? Is it possible to remove all of the calculus by routine scaling or should the root be planed by thorough curettage? How much calculus and cementum remains after the root is planed by curettes until it feels smooth and hard? What is the microscopic appearance of a root that feels smooth and hard either to a curette or an explorer? Do the effects of routine scaling and routine planing differ histologically?

Previously, some light has been shed on a few of these questions. Zander described four methods of calculus attachment to the root surfaces.[6] The calculus was attached in total or in part to the secondary cuticle, to the surface of the cementum, in the cementum by an actual penetration of the bacteria as part of the organic matrix, and finally in resorption areas in the cementum. Two of his photomicrographs demonstrated calculus penetrating the cementum, filling a resorption area that extended through the cementum to the dentine, and spreading laterally along the dentino-cemental junction. From his work, it seems that in order to remove all the calculus, it may be necessary to remove part of the cementum and in some instances all of it.

After extractions, Riffle studied the effects of extraoral curettage on the roots of teeth.[7,8,9] He states that the results were the same whether periodontal curettes or spoon-shaped vulcanite scrapers were used. From measurements before and after curettage, he concluded that the cementum was removed in its entirety in the region of the cemento-enamel junction. He also stated that planing periodontal pocket areas resulted in the removal of all of the cementum. Riffle concluded that it was impossible to remove only one-third of the cementum as some periodontists advocate. He did not study the teeth histologically after curettage. Naturally, one must ask whether it can be concluded that all of the cementum was removed without a histological study. Not only is the preceding question raised, but also one might ask whether it can be assumed that the results of planing the roots of extracted teeth are the same as root planing while the teeth are in the mouth.

Because many of the questions listed above were unanswered, it seemed that further investigation of the effects of curettage on the root surfaces was desirable. In order to answer some of the above questions and to increase our knowledge of root curettage, teeth were curetted prior to extraction and the effects were evaluated with a dissecting microscope and with a microscopic study of histologic section.

PROCEDURE

A total of eighteen teeth, previously condemned for prosthetic reasons, were selected for this study. They included both anterior and posterior teeth, and their gingival sulci varied in depth from two to seven millimeters. All instrumentation was done prior to extraction of the teeth. The instruments used were the Gracey and McCall curettes. Six teeth were scaled

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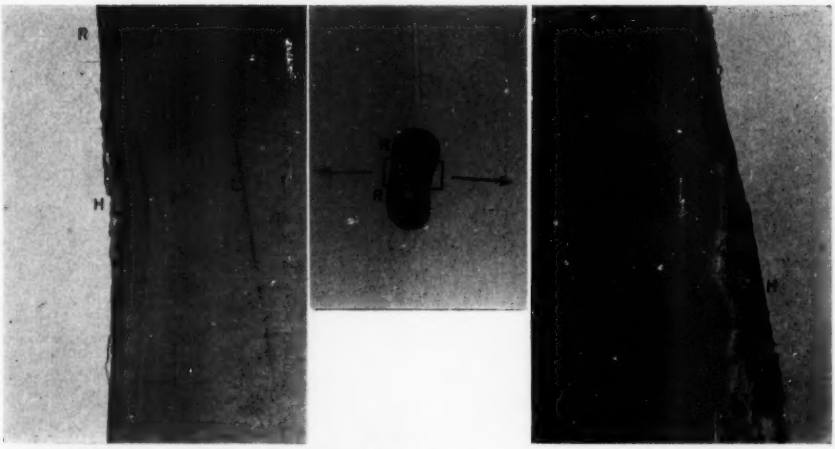


Fig. 1. Calculus (H) remained on the mesial and distal surfaces after routine scaling. Some of the outer layers of cementum were removed (R).

routinely with curettes to remove the calculus, while twelve were curetted to remove the calculus and to plane the root surfaces. The roots of the latter teeth were planed with curettes until they felt smooth and hard to a curette and a number seventeen explorer.

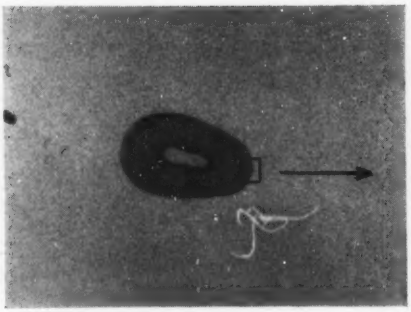
Upon extracting the teeth, they were studied with a dissecting microscope. After this, they were fixed either in formalin or Zenker's solution, decalcified with trichloroacetic acid or with nitric acid, embedded in celloidin, sectioned serially, and stained with hematoxylin and eosin. Some of the sections were also stained with a tissue Gram stain. All of the teeth were cross sectioned.

RESULTS

On all six teeth that were routinely scaled and not planed, calculus was remaining on many of the root surfaces. The calculus remaining varied in amount and thickness. In some instances, it was evident that the superficial layers of calculus had been removed, but the deeper layers were still attached to the teeth. On a few surfaces, the calculus was intact and was thick (Fig. 1). On one surface of a tooth, calculus was still remaining in an irregularity of the cementum even though some of the superficial layers of the cementum adjacent to this area had been removed (Fig. 1). Calculus was observed both in the areas of the cemento-enamel junction and the periodontal pocket. Sometimes it was near the bottom of the pockets, and other times it was near their lateral borders.

A few surfaces demonstrated that a small amount of the outer layers of cementum was removed with a curette (Fig. 1). In two sections of one tooth all the cementum had been removed. It was not removed completely from the other teeth. The roots of the teeth were not smooth after routine scaling.

Of the twelve teeth that were thoroughly curetted to remove the calculus and plane the root surfaces, only one had calculus on one of its surfaces. It was a small amount, and part of it was lying in a cemental defect. The cementum had been curetted on each side of the calculus, and some of the outer layers of the calculus and cementum was removed. A short distance from the calculus, the cementum had been totally removed. Upon studying the histological



sections, one could see that the calculus and cementum in this area might feel smooth to a curette or an explorer (Fig. 2).

In all the teeth, the cementum had been entirely removed in most of the pocket areas and in the region of the cemento-enamel junction (Fig. 3). Where cementum remained in the pocket area, there was only a small thin layer covering the dentin (Fig. 4). It could be clearly seen that the outer layers had been removed. Whenever cementum remained, the root felt smooth and hard. Microscopically the cementum usually appeared to be smooth (Fig. 4, 5). On some portions of the planed roots a few small islands or spots of cementum remained, while on others a single piece of cementum remained which was larger in area but very thin (Fig. 5). Wherever the cementum was completely removed, the exposed dentine felt smooth and hard to touch, and microscopically it appeared to be generally smooth (Figs. 3 and 5). The cementum adjacent to the bottom of the pocket was intact and the sections below this level demonstrated connective tissue fibers embedded in the cementum. Occasionally, epithelial cells were adherent to the cementum. Although the planed surfaces felt smooth and hard to a curette, they were not always perfectly smooth when studied with the dissecting microscope. This infrequent finding was verified in the histologic sections which revealed a few microscopic irregularities. In resorption areas of three teeth, secondary cementum was observed. The matrix of an

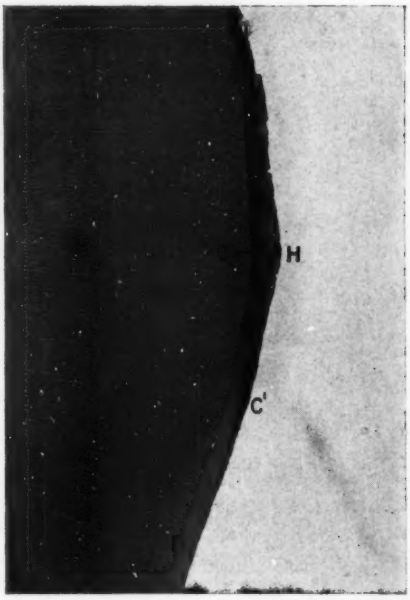


Fig. 2. Generally the root of this tooth was smooth, but calculus (H) remained after routine planing. Some of the superficial layers of the calculus appear to be removed but the deep layer remains. The cementum (C) was curetted adjacent to the calculus at C'. All the cementum was removed exposing the dentine (D) a moderate distance from the calculus.

enamel pearl was present in the bifurcations of the four of the nine molars (Fig. 6).

DISCUSSION

In this study, it was apparent that routine scaling did not remove all the calculus from the root surfaces. In order to remove all of the calculus, it was necessary to thoroughly plane the root surfaces in the subgingival areas. The planing removed all the calculus with one exception. In this instance, part of the calculus was lying in an irregularity of the cementum. This surface felt smooth to a curette and an explorer, and upon microscopic examination, it was evident that the calculus had been curetted smooth along with the cementum. A short distance away from this area and on each side of it, the cementum had been completely removed down to the dentine.

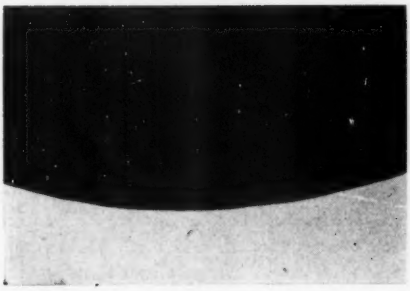


Fig. 3. Cross section of a planed root, all of the cementum was removed and the surface of the dentine (D) is smooth.

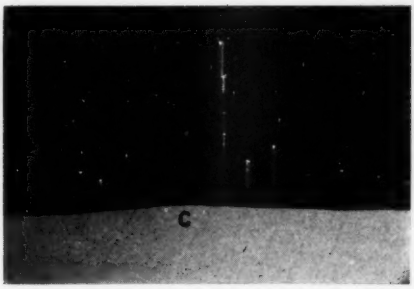


Fig. 5. Cross section of a planed root, a small portion of cementum (C) remained. It is thin and smooth. On both sides of the cementum (C), the dentine (D) was exposed and smooth.

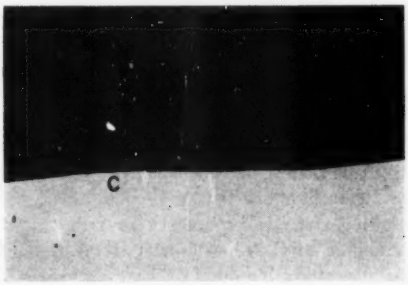


Fig. 4. Cross section of a planed root, the outer layers of cementum were removed and the remaining cementum (C) is very thin and smooth.

From Zander's investigation[6] and this survey, it seems evident that in order to remove all the calculus, it is necessary to remove part of the cementum and in some instances to remove all of it.

It was demonstrated microscopically, that a smooth hard surface of a root, can be either one with all of the cementum removed and the dentine exposed or one with some cementum remaining on the dentine. In the twelve teeth that were planed, the dentine was exposed in over one-half of the total area from the cemento-enamel junction to the bottom of the pocket. It is necessary to remove some of the cementum or all of it to obtain a smooth hard surface. Some of these findings tend to verify Riffle's investigation in which he planed extracted teeth extraorally with curettes and vulcanite scrapers.[7, 8, 9] However, one must disagree with his conclusion that it is necessary to remove all of the cementum to get a smooth hard surface. In some instances, a thin layer of cementum remained, and it felt hard and smooth.

After attaining a hard, smooth surface, it was impossible to determine by touch whether or not the cementum was entirely removed. It was impossible to predict the amount of cementum remaining without studying the microscopic sections. Both cementum and dentine manifested a few microscopic irregularities even though it felt smooth.

It was interesting to see that routine scaling, in an attempt to remove all the calculus, failed in all instances. Although all the calculus was not removed, a small part of the adjacent superficial layers of cementum was removed in a few instances. Not only were small amounts of cementum removed, but also in some instances the superficial layers of calculus were removed while the deeper layers remained.

Although no attempt was made to remove only one-third of the cementum as proposed by some periodontists, it was impossible to predict how much of the cementum had been removed clinically until the teeth were studied microscopically. If all of the cementum is removed, it can be demonstrated histologically. However, it was impossible to determine histologically whether one had removed one-third, one-fourth, or one-half of the cementum. The reason is that it was not possible to de-

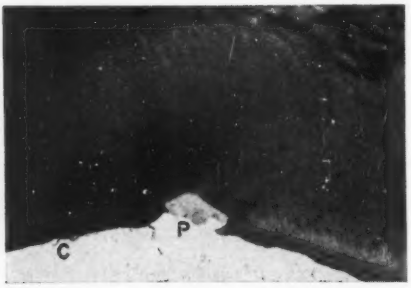


Fig. 6. Cross section of a root apical to the bottom of the pocket, a few connective tissue fibers remained on the surface of the cementum (C). The matrix of an enamel pearl (P) is present on the surface of the dentine (D) and in a break in the continuity of the cementum.

termine the thickness of the cementum before curettage.

From this survey, it appears that thoroughness in root planing cannot be overemphasized. Every surface of every tooth must be meticulously planed to remove all the calculus and to insure a smooth hard root surface.

SUMMARY AND CONCLUSIONS

Prior to the extraction of eighteen teeth, intraoral root curettage was completed. Routine scaling to remove only the calculus was done on six of the teeth and thorough root planing was completed on the other twelve. The teeth were studied with a dissecting scope, and with a microscope after preparing histological sections.

After routine scaling, calculus remained on some surfaces of all six teeth. On a few surfaces whether the calculus was removed completely or partially, the superficial layers of cementum were curetted. The root surfaces were usually rough.

After thorough planing with a curette, all the calculus on all surfaces of eleven of the teeth was removed, but on one surface of the twelfth tooth calculus remained and part of it was in a cementum irregularity.

In the subgingival areas of twelve teeth, the cementum was curetted and removed totally or partially. In over one-half of the total subgingival area, all the cementum was removed exposing the dentine. Whenever cementum remained, it was very thin, and it felt smooth and hard to a curette or an explorer. Microscopically, it usually appeared to be smooth. When dentine was exposed, it too felt smooth and hard to either a curette or an explorer, and it appeared to be microscopically smooth in most instances. The cementum and dentine felt equally smooth and hard. However, a few microscopic irregularities were seen.

It was impossible to determine by touch whether one was curetting cementum or dentine. Although it was the usual microscopic finding that all of the cementum had been removed, it was impossible to determine by the sense of touch whether or not cementum remained. Hence, it would be impossible to remove one-third of the cementum.

From this study, it seemed evident that routine scaling was not adequate to remove all the calculus. Thorough planing of the root surface is imperative to remove the calculus and to produce a smooth, hard root surface. To obtain a root surface free of calculus, some of the cementum must be removed, and in some instances all of it.

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Clinical Procedures in Periodontal Therapy*

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IN the treatment planning of periodontal cases, the importance of a differential prognosis is not always given sufficient attention. This is based on the functional requirements and the "life expectancy" of the teeth following periodontal therapy.

The projected plan of treatment should include not only the elimination of the tissue pathology but consideration of other measures which will contribute to the successful maintenance of the teeth. Practically every dental procedure either directly or indirectly involves the periodontal tissues. The interrelationship between periodontal disease and restorative dentistry is an established fact but so frequently overlooked or ignored. Since faulty dentistry is an etiologic factor in the inflammatory reaction it is axiomatic that proper restorative procedures are essential to the physiologic response of the periodontal tissues. Periodontal cases present so many different but related factors that consultation with the patient's dentist is important. A brief description of several of these problems will show the necessity for modification of the prognosis and where periodontal therapy must be integrated with other branches of dentistry.

Teeth with single roots and advanced bone loss may be favorable for periodontal therapy when subjected to only the normal stresses of mastication but when in addition they are required as abutments, the prognosis may be poor. In many cases of teeth with mobility the use of a fixed splint would aid in their retention.

There are those cases with generalized and extensive periodontal involvement where the extraction of all remaining teeth in the arch would be the ideal procedure from a periodontal standpoint. But frequently there is a psychological problem, especially with women patients, and their dentist may feel that it is not advisable to put them into a full denture immediately. Some of the least involved teeth are retained for awhile to support a partial appliance. They will require periodontal treatment until such time as a full denture is unavoidable and the patient in a better state of mind about the situation. Since the conservation of the alveolar bone is all important, the periodontist should keep these cases under close observation both clinically and radiographically.

Another type of case is one having a probable periodontosis background which on examination shows migration and mobility of the teeth, deep pockets, no appreciable calculus, good mouth care and no marked inflammation of the gingival tissues. The upper teeth are usually affected to a greater degree than the lower teeth, with mobility in the bicuspid areas, migration in the anterior region and involvement of the molar trifurcations. Other factors having a predisposing influence would be the length and shape of the roots and the type of alveolar trabeculation. In those cases where fixed splinting is not advisable, it is difficult to obtain and maintain a balanced occlusion. Under these conditions, the teeth are unable to withstand the forces of mastication following periodontal therapy and probably some prosthesis. Progressive mobility of these upper teeth occurs with inflammation of the periodontal tissues and concurrently the lower teeth become further involved in the overall process. In the treatment planning of this particular type of case, the periodontist may recommend the extraction of all the upper teeth. This reduces the occlusal forces on the lower teeth since they would be in occlusion with a full upper denture. The prognosis for perio-

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dontal therapy of the lower teeth therefore is more favorable under these conditions. This may seem as a radical procedure to the referring dentist and requires careful explanation of the problem to show him that it is a conservative measure. Several cases treated in this manner have been under observation from three to eleven years and the results in retaining the lower teeth have been most satisfactory. In two cases where this procedure was not followed because the patient refused to have the extractions, there was progressive mobility and recurrent inflammation. Periodontal therapy was not successful and within four years it was necessary to also extract the lower teeth. The problem then was retention of the lower denture because of the generalized loss of alveolar bone.

Traumatogenic occlusion is most frequently corrected by stoning the interfering cusps or cuspal inclines but in many cases it is necessary to replace or build up the occlusal surfaces in order to obtain proper functional balance. Crowding or buckling of the incisor teeth may be corrected by extraction of the most malposed tooth and realignment of the adjacent teeth by orthodontic procedures. Wherever possible, diastemas should be closed with restorations. Migration of the anterior teeth requires orthodontic measures and either a retainer or fixed splinting to hold them in position.

These are some of the problems which must be considered in the treatment planning of periodontal cases. Their solution requires not only proper diagnosis and periodontal therapy but consideration of the functional, mechanical and often psychological factors in order to maintain the teeth and investing tissues in a physiologic state of health.

A treatment planning clinic has been set up at the University of California Dental School to assist the students in planning the overall treatment of their complex periodontal cases. This teaches them the more general and proper concept of periodontal therapy and the importance of planning restorative procedures in relation to their influence on the periodontal tissues.

The periodontist who has an understanding of the basic principles of balanced occlusion, correct operative and prosthetic procedures has an excellent opportunity to make suggestions to the patient's dentist. When presented to him in a tactful manner, there is seldom any questions about his complete cooperation.

Some of us with years of experience still do not know all the answers. There may be a similarity of symptoms but there is a dissimilarity in cases. Therefore consultation and collaboration between the periodontist and the referring dentist is important in all cases. There is a mutual understanding of each others responsibility and more successful periodontal therapy. It would also result in better dentistry, and an increased knowledge of and more interest in periodontia on the part of the general practitioner. Many of them have had no training in periodontia and discussion of treatment planning with the periodontist will stimulate a much earlier recognition of the clinical symptoms and interpretation of the radiographic evidence of periodontal disease.

OBJECTIVE OF THERAPY

The objective of therapy is to stop the progress of the disease and to maintain the teeth and their investing tissues in health and function for as long a period as possible. This should be brought about by procedures which will resolve the inflammatory process without unnecessary loss of soft tissue or alveolar bone, and reduce the depth of the pocket to as shallow a sulcus as possible. Some periodontists state, that following therapy, the depth of the sulcus should be 0 to 1 mm., which incidentally, does not always occur, due to the many variables that pertain to each root surface as well as each individual patient. Other clinicians are of the opinion that where tissue attachment is not obtained, the unattached tissue can frequently become so closely adapted to the root surface as to

eliminate a macroscopic sulcus. There are cases where this close adaptation does not occur and residual sulcus of 2 or 3 mm. is the best result that can be obtained. Orban states, "One must differentiate between a sulcus and a pocket. If the symptoms of inflammation are absent, the area between the tooth and the gingiva is a sulcus, regardless of its depth. If the gingival tissues are inflamed, the area is a pocket no matter whether it is shallow or deep." The question which must be decided in the operator's mind is whether he will accept this condition of a moderately deep sulcus without clinical symptoms of pathology, as a satisfactory response to treatment or whether this sulcus should be eliminated by resection. A deep sulcus certainly has a greater potential of developing subsequently into a pathologic pocket than a shallow one, but this possibility is lessened by adequate supportive care, i.e., periodic prophylaxis and proper oral hygiene. While this may not be the ideal condition, yet clinical observation of many such areas has shown that they can be maintained for years without recurrence of inflammation or further resorption of bone.

There are several different clinical procedures advocated for treating periodontal disease, including subgingival curettage, gingival resection, electrosurgery and chemosurgery. This discussion will be confined to curettage and resection, since they are most frequently used in periodontal therapy.

The type of local treatment selected depends on the character of the lesion about the individual teeth and on the overall diagnosis. In many cases the results obtained by subgingival curettage are such that it is the only procedure necessary. In some cases, initial supra and subgingival scaling is done to remove the local irritating factors, thereby reducing the inflammatory process and improving tissue resistance before performing surgery. In other cases, gingival resection is the initial method of treatment that is indicated. The lesions often vary in the same mouth and consequently may require a combination of different procedures. All methods of therapy have advantages and disadvantages and only through a knowledge of the histopathology and the tissue healing potential can sound judgment be developed.

SUBGINGIVAL CURETTAGE

Menkin[2] states, "Inflammation is a manifestation of a severe cellular injury. . . . . Cellular injury by an irritant alters the internal cell biochemistry with the liberation of various common denominators which reasonably explain some of the biological manifestations of inflammation." An irritant may be defined as any agent of a physical, chemical, thermal or bacterial nature that interrupts normal cellular metabolism. The inflammatory process can be reduced by removal of the irritating factors. Therefore, the rationale of subgingival curettage—the establishment of favorable conditions which enable the periodontal tissues to repair the lesion. This is accomplished by removal of the local irritation, curettage of the surfaces of the tissues forming the pocket and retention of the blood clot.

Treatment—In subgingival curettage, the modified Younger curettes are preferred. They are very thin and may be inserted into deep pockets with comparative ease and with a minimum amount of tissue trauma. Cone-socket handles are used to facilitate more rapid and easier replacement when the instruments become worn and their light weight helps to transmit a better sense of touch to the fingers of the operator. The curvature of the shank and blade of these instruments are such that they have a universal application. The rounded cutting edges of these curettes are best adapted to the root surfaces. The deposits of calculus may be removed and all root surfaces more smoothly curetted than with the hoe type, especially in pockets having a narrow orifice.

The first part of the therapy consists of removing the debris and supragingival calculus. This is accomplished with curettes (#5 and #6) (Fig. 1) which have a

thicker shank and blade than that used for subgingival scaling. The scaler is allowed to follow the calculus apically until it engages the edge of the mass at the gingival margin and is then brought incisally or occlusally. A short sickle is often very useful in the interproximal areas. Since repeated strokes are necessary, care should be taken not to unnecessarily lacerate the gingival tissues. The instruments are held with the pen grasp, i.e., between the thumb, index and third fingers. It is essential to have definite and firm finger rests on the incisal and occlusal surfaces for proper manipulation and control of the curettes.

The next step is treatment of the pathologic pocket and the basic set of instruments (Fig. 1) consists of six right and left curettes (#1 and #2, #3 and #4, #7 and #8) and a long thin sickle (#13). They must always be kept very sharp since they have the dual purpose of removing the subgingival calculus and curetting the epithelial tissue. The curettes are carefully inserted into the pocket and manipulated with a lateral stroke—backward and forward—removing the deposits and planing the cemental surface. They can also be used with a push or pull stroke or in all directions according to conditions. The curettes should be held very lightly with the shank close to the crown, parallel to the long axis of the tooth and the cutting edge of the blade at about a 45 degree angle to the root surface. Coincidently as one edge of the curette engages the cemental surface, the opposite edge is in contact with the soft tissue wall of the pocket. As the curettage progresses, pieces of granulomatous tissue and undoubtedly epithelium are brought to the surface. In some types of pockets a special effort is made to curett the epithelial lining. Whether or not all the epithelial cells are completely removed is a matter of conjecture from a clinical standpoint. The excellent response of the tissues to this therapy, especially in intrabony pockets where deposition of new bone frequently occurs, is an indication that favorable conditions for tissue repair are obtained.

In general, the long shank curettes (#1 and #2) are used in the molar regions and in deep pockets, the short thin instruments (#3 and #4) in shallow pockets, bifurcations and moderately deep pockets around bicuspids. The very short curettes (#7 and #8) are used for small pieces of calculus adjacent to the gingival margin. The long sickle is a fine sharp finishing instrument and universally used. Files and the Gracey set of scalers are frequently used as adjuncts to the Younger curettes.

The curettage of the cementum is best accomplished by carefully following the contour of the root surface with a stroke of medium length and in a smooth, rhythmic and continuous manner. A long stroke, or short and jerky movements of the instruments should be avoided as they could cause unnecessary laceration of the soft tissues and may produce deep cuts in the cementum. It is very important to reach the bottom of the pocket around the entire periphery of each tooth. Since there is a layer of epithelium and connective tissue between the base of the pocket and the alveolar bone, the bottom of the pocket is determined clinically by one's tactile sense and not by the radiographs. The pockets should be treated in a methodical manner, commencing at some definite point and proceeding from tooth to tooth until all have been treated. Unless conditions dictate otherwise, personal preference is to begin in the lower anterior region. The curettage around one or two deep pockets or a group of several shallow pockets should be completed before proceeding to the adjacent area and only as many pockets should be treated as can be finished at that appointment. In the interdental areas this would also include both the mesial and distal surfaces of the teeth and the strokes of the curette should cross from labial and buccal to lingual to insure thorough curettment. An explorer or other suitable instrument is used on completion of the curettage to detect any roughness of the root surface. The depth of the pockets are measured with a periodontometer and recorded for future reference.

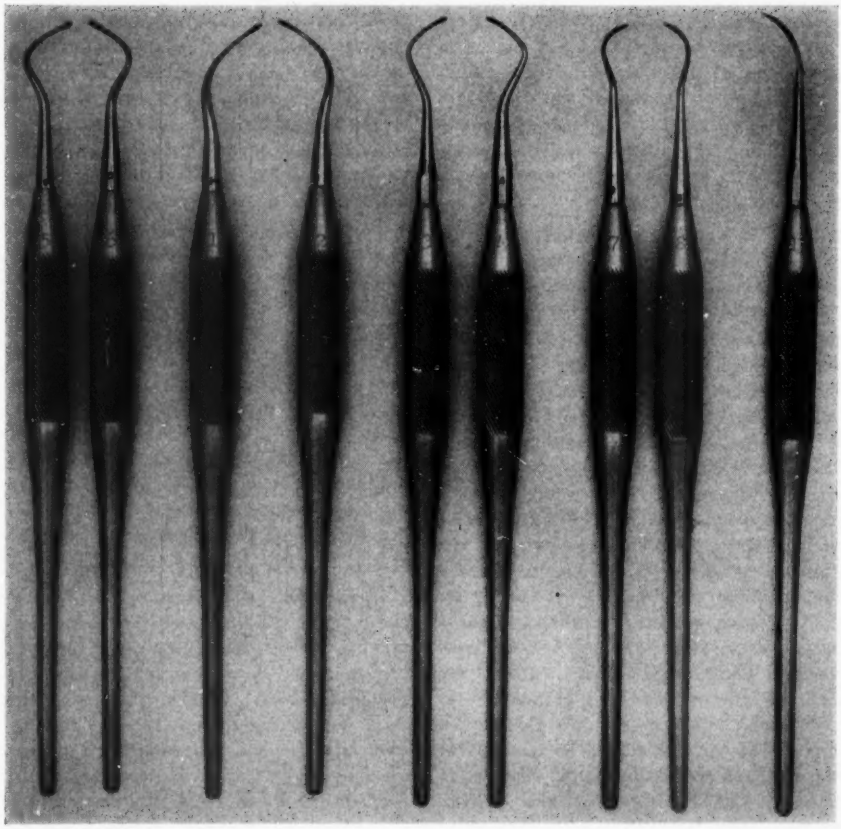

Fig. 1. Basic set of instruments used for subgingival curettage.

The final step in subgingival curettage is to leave a clean wound and establish a blood clot. In many cases, it is an advantage to protect the curetted area with a surgical cement dressing. In this instance, it is not used as a pack in gingivectomy, but mixed to a thinner consistency and carefully applied so that the clot will not be disturbed. This dressing should be maintained for at least one week or longer if indicated. The scaling and curettage in pockets of moderate depth can frequently be done without undue discomfort or pain to the patient, but there are cases where a topical or injected anaesthetic is necessary to accomplish the objective of therapy. Effective subgingival curettage depends on the thoroughness with which it is performed. The radiographs should be used during treatment for correlation with the clinical conditions as to the location, type and approximate depth of the pocket.

GINGIVAL RESECTION

There are some conditions of the gingival tissues and some types of pockets which are unfavorable for subgingival curettage and excision of all or part of the involved gingival tissue is necessary. The surgical technics which might be used include, gingivectomy, papillectomy and gingivoplasty.

Gingivectomy—Gingivectomy is the removal of the detached gingival tissues to the bottom of the pocket in order to eliminate the soft tissue side of the pathologic pocket. The principal items of the armamentarium include gingivectomy knives, a small pair of scissors, spoon curetts, and a zinc oxide-eugenol dressing.

The first step is to obtain anaesthesia of the area to be operated. The depth of the pockets is then determined and with a periodontal probe, explorer, or other pointed instrument, small punctures are made on the facial and lingual tissues at the proper distance from the gingival margin to indicate the base of each pocket. The incision is made in a continuous manner along a line slightly apical to the puncture points, thereby following the bottom of the pocket on both the facial and lingual surfaces so as to produce a satisfactory bevel of the operated gingivae. The loose tissues are removed and the bleeding controlled by packing with gauze—in the interproximal areas the use of gauze twills is more convenient. The resected area is then carefully examined for any remaining granulomatous tissue, which is removed with curetts. All deposits of calculus should be removed at this time and the surfaces of the cementum, especially at the base of the pocket, thoroughly planed with curettes or files.

The incision should be deftly made with very sharp knives, leaving no ragged edges or tags. Should such occur, they may be removed with a pair of suitable scissors. The next step is to allow a blood clot to be established. A surgical cement dressing is then carefully applied over the entire wound surface with only sufficient pressure for retention and without disturbing the blood clot. The material should be mixed to a consistency which allows it to be easily rolled between the fingers. This dressing provides a protective covering for the tissues during the healing period. It should be removed in about seven days, the area irrigated with warm water or a saline solution, and the surface of the wound and adjacent tissue cleaned with cotton saturated in a weak solution of hydrogen peroxide. Any excessive proliferating granulation tissue on the wound surface is treated with trichloracetic acid. The dressing is replaced and should be maintained for at least 10 days or until such time as the epithelium has covered the wound. The patient must be cautioned not to do anything which might disturb the dressing, such as brushing the area or eating on that side of the mouth. Careful brushing of the remainder of the teeth should, of course, be continued. Proper oral hygiene of the operated area should be instituted on removal of the dressing and daily use of the interdental stimulator should be stressed. Resection of acutely inflamed gingival tissue is contra-indicated because of the accumulation of bacteria and toxic products. In such cases, the procedure is to remove the local irritating factors by scaling and reduce the inflammation as much as possible prior to the gingivectomy.

Papillectomy—Papillectomy is the excision of the interdental papilla, without involving the marginal gingivae. This procedure is used in areas where the papilla is detached, fibrotic, or edematous, or where the inflammation has not been reduced by removal of the local irritation. Such papillae are frequently irritated by impingement of food during mastication. This loose tissue also offers an excellent opportunity for accumulation of debris between it and the tooth surface. They are frequently hemorrhagic and bleeding occurs with but slight pressure from the toothbrush. The line of incision should be elliptical especially in the anterior region and at a forty-five degree angle, the purpose being to form an inclined plane and interproximal sluiceway for the excursions of food. The surgical cement dressing in this instance should be of stiff consistency and firmly packed into the interproximal space. The post operative care is the same as for gingivectomy. A combination of papillectomy and subgingival curettage is occasionally used in treatment of deep intrabony pockets.

Gingivoplasty—Gingivoplasty is the reshaping of any abnormal contour of the gingivae so that a better architectural form

may be obtained. This technic is used in areas where the normal festooning of the gingival margin has been lost and a thickened shelflike condition has developed, and also in cases of necrotizing ulcerative gingivitis where following treatment, the interdental gingivae have a cup-shaped or crater formation. These improper anatomical conformations have a tendency to retain food debris and subsequent gingival inflammation will occur. Since no deep periodontal pocket is present, the incision is usually shallow with only the surface tissue removed. It should also be made at an angle or beveled to obtain more ideal tissue contour and restore a thin knifelike gingival margin. A surgical dressing is applied when several adjacent areas are treated and the usual post-operative measures are followed. Electrosurgery has been used very successfully in gingivoplasty and some operators prefer this technic instead of the incision.

A discussion of therapy would not be complete without mentioning that there are several other surgical procedures, such as the flap operation, the modified flap technic and the semilunar flap. They may be indicated in specific instances where removal of the gingiva to the bottom of the pocket is contraindicated for aesthetics, architectural and other reasons. Osseous resection is another periodontal procedure advocated by some periodontists for reshaping the crest of the alveolar bone in intrabony pockets and molar bifurcations.

HOME CARE

Thorough instruction in proper home care is a very important clinical procedure. Observation has shown that the majority of periodontal patients do not know how to properly care for their mouths because they have received no instruction from their general dentist. The selection of a toothbrush should be made on the basis of an analysis of the objective—namely the removal of the soft debris adhering to the surfaces of the teeth and the development and maintenance of good gingival tissue tone. A satisfactory type of brush is one having many tufts with small diameter bristles of moderate length and a flat or level trimmed brushing surface—the so-called straight toothbrush.[3] The many bristles will cover more area of the tooth surface, thus producing a more effective cleaning result and with less danger of traumatizing the gingival margin. Good mouth hygiene means not only the correct use of the toothbrush but also thorough care of the interproximal areas. The agents used for this purpose are dental floss, dental tape, soft wood points, and soft rubber cones. Each has its particular indication, depending on the height of the septal tissue or size of the interproximal space and should be suitably prescribed by the patient's dentist. The cleaning of the tongue should also be recommended as a daily procedure and the type of brush just mentioned is an excellent agent for this purpose. The principles of correct mouth care should be discussed with the patient and the most effective method of instruction is to take the toothbrush, dental floss, soft wood points or interdental stimulator and actually demonstrate their use in the patient's mouth. Repeated demonstrations are often necessary before they learn to do it correctly. The patient should not be dismissed until he has shown his ability to properly care for his teeth and investing tissues. The success or failure of periodontal therapy depends not only on the good judgment and ability of the operator but also on the cooperation of the patient. The significance of this fact must be fully emphasized to the patient. Effective home care is a therapeutic procedure equal in importance to that of treatment of the periodontal lesion.

There are other factors which must also be considered in periodontal therapy, such as relief of excessive stress and placing the teeth in the best possible occlusal balance; the stabilization of mobile teeth with temporary or permanent splints; the investigation of any nutritional deficiencies, nervous habits, and psychogenic problems. The physical condition of the patient must also be considered since it is often a predisposing or contributing factor in producing

degenerative changes in the alveolar bone or in lowering gingival tissue resistance. The correction or elimination of these conditions will result in obtaining a more satisfactory response of the tissues to subgingival curettage and gingival resection.

SOME INDICATIONS FOR SPECIFIC THERAPY

It is not a matter of one method being superior to another but which procedure will reduce the inflammation, restore the tissues to physiologic health and insure the maximum amount of function. In many periodontal pockets, subgingival curettage is the procedure that should first be instituted. Subsequently, if there are pockets that do not respond as anticipated after curettage and there is a persistence of the inflammation, then resection of the gingival tissue is necessary.

There are some specific conditions where subgingival curettage is contra-indicated and gingival resection is the method of treatment that should be first employed. For example:

1. Loose gingival flaps and pedunculated papillae.

2. In cases of periodontosis showing migration of the teeth, deep pocket formation and loss of bone.

3. Where there is extensive destruction of both the interproximal soft tissue and bone, such as often found in cases having had recurrent necrotizing ulcerative gingivitis. In these cases, the height of the facial and lingual gingival wall is reduced to the bottom of the interproximal crater to enable better home care of the area.

4. In those pockets where the gingival tissue is thin with no marked inflammatory reaction and where there is an absence of irritating factors, such as calculus.

5. Areas of fibrotic gingival hyperplasia where prophylaxis, curettage, toothbrushing and interproximal stimulation will not reduce the tissue to normal contour.

These specific conditions are generally not found throughout the entire mouth but usually only around several teeth. Therefore, periodontal cases are frequently treated by a combination of the different procedures. One must generalize somewhat in outlining the indications for therapy as there are always individual problems which must be considered, such as the patient, the clinical conditions and the experience of the operator.

Cases have been observed where the gingival tissues have been resected regardless of the etiologic factors, the status of the gingivae, or the depth of the pockets. One reason for this situation is the overemphasis which has been given to the removal of all detached tissue. This undoubtedly has led many to assume that resection is the indicated and most satisfactory procedure in every case. Consequently, there are many cases of simple gingivitis which are treated by gingivectomy, where a thorough scaling or prophylaxis would have reduced the inflammation and corrected the condition.

SUMMARY

1. The importance of a differential prognosis and consultation with the patient's dentist is discussed.

2. The clinical procedures in periodontal therapy including subgingival curettage, gingivectomy, papillectomy and gingivoplasty are described.

3. Patient instruction in proper toothbrushing and interproximal care is an important clinical procedure.

4. Several specific periodontal conditions where subgingival curettage is contra-indicated are enumerated.

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The Charters Method of Toothbrush Massage: *A Valuable Supplement in Periodontal Therapy*

by HARRY ROTH, D.D.S.;* JACK THALLER, D.D.S.,† AND NORMAN E. ALDERMAN, D.D.S.,‡
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TOOTHBRUSH massage is accepted by most general practitioners and periodontists to be of cardinal importance in maintaining oral health. Toothbrush massage implies the placement of the lateral surfaces of the toothbrush bristles firmly against the gingivae and assumes a greater value than cleansing action alone.

Proper toothbrush massage should accomplish the following:[1]

1. Creation of firm gingival tissue, resistant to disease.
2. Stimulation of supporting structures.
3. Cleansing of the teeth (removal of debris and soft stain).
4. Prevention of caries.

Benefits derived from massage:[2]

1. Periodontal stimulation
2. Improved periodontal circulation
3. Decrease of gingival bleeding
4. Improved gingival color and tone
5. Resistance of gingivae to infection and trauma
6. Toughening and tightening of epithelial collar
7. Reduction of gingival sensitiveness
8. Lessening of deposits on teeth
9. Decrease of caries
10. Improved pupal circulation
11. Lessening of root sensitiveness
12. Insurance of patient cooperation

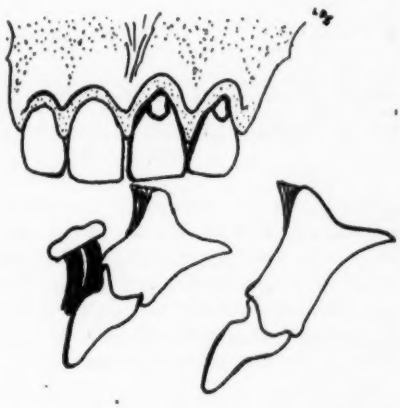

Fig. 1. The bristles in the Charters method engage the eroded areas and keep the surfaces free of accumulated food particles which tend to lessen the susceptibility to caries.

Fig. 2. The attached frenum is one of the factors causing recession of the gingivae. Teeth in malposition in the arch are subject to food impactions and are cleansed with difficulty. The Charters method (acting as many interdental stimulators working simultaneously) lends itself more readily in creating a more favorable topography.

PURPOSE

Because patients' dentitions vary con-

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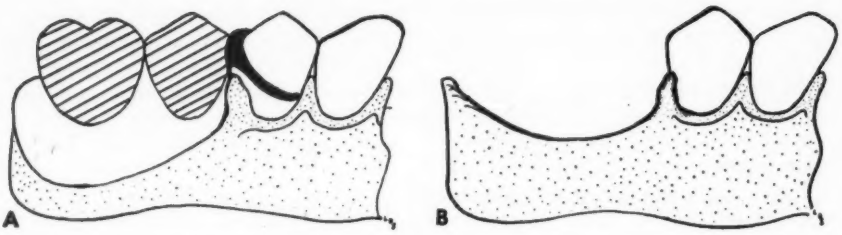

Fig. 3a. The gingival tissue adjacent to the clasped tooth invariably becomes enlarged due to the pressure and the movement of the saddle impinging upon the tissue. The Charters method is indicated here.

Fig. 3b. Gingival enlargement more demonstra ble with the removal of the partial denture.

siderably, no one method of toothbrush massage can serve all. The dentist must observe each case individually, noting the variations in the patients' oral architecture; and then prescribe the particular method or methods of toothbrush massage best suited to the case under treatment.

METHOD

Patients were selected from those treated in the Periodontia Clinic of New York University College of Dentistry, by Junior and Senior dental students. The treatment consisted of scaling, toothbrush demonstration, diet evaluation and correction, temporary splinting when indicated, occlusal equilibration, subgingival curettage, portepolishing, habit correction, systemic evaluation and correction when necessary, and gingivectomy and gingivoplasty when required. The result of the treatment was satisfactory in most cases. However, it was noted that in some instances isolated areas failed to respond with the routine home care procedure employed. For these regions, an attempt was made to supplement the routine modified Stillman toothbrush procedure and effect a more favorable result by the Charters method. This method of toothbrush massage was employed on the labial only. The rationale for the use of Charters method of brushing is to enable the sides of the bristles to work interproximally and to cleanse the embrasures as well as to reach the thick labiolingual plate of gingival tissue for massage. This perhaps can best be described as many interdental stimulators working simultaneously. The modified Stillman technic does not afford adequate contact of the bristles with the interproximal tissue when the interproximate papillae are not intact.

Charters Method: The ends of the bristles are placed in contact with the enamel of

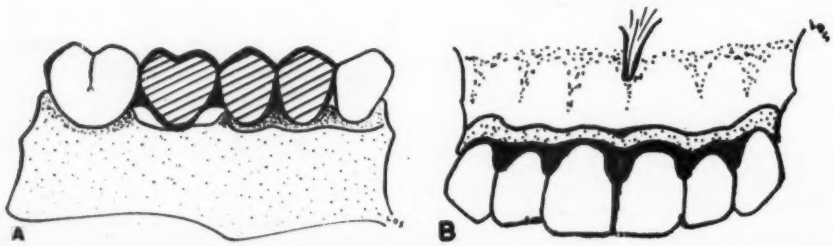

Fig. 4a. Soldered contacts placed too far gingivally in fixed bridges result in narrow embrasures and meager self-cleansing areas.

Fig. 4b. Food particles are inevitably trapped in the interproximal embrasures with resultant inflammation and overgrowth of the tissue.

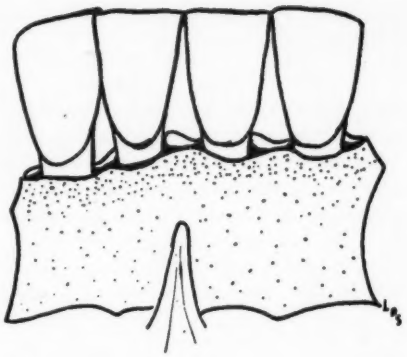

Fig. 5. The widened interproximal tissue from labial to lingual needs proper stimulation and cleansing. The Charters method of brushing fulfills the requirements of ultimate treatment subsequent to the surgical procedure.

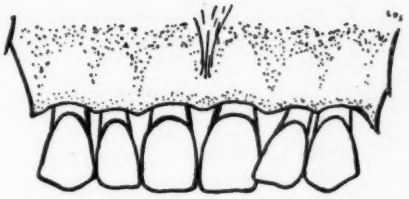

Fig. 7. Subsequent to recurrent episodes of acute necrotizing ulcerative gingivitis, the interproximal craterization created by the sloughing of the interproximal tissue leaves high labial and lingual flaps of tissue. The center of the former papillae is below the crest of the outer walls. The Charters method of brushing is most desirable in such areas.

the teeth and the gingival tissue with the bristles pointed at about a 45° angle toward the occlusal surfaces. Lateral and downward pressure is then placed upon the brush which is vibrated gently with the handle being moved back and forth a millimeter or so. This gentle vibratory procedure forces the ends of the bristles between the teeth and cleans the interproximal tooth surfaces while it massages the gingiva.

INDICATIONS

Gingival conditions found to respond more favorably to the Charters method are as follows:

1. Teeth with cervical erosion
2. Teeth with recession
3. Malposed teeth
4. Abutment teeth supporting a removable prosthesis
5. Diminished vestibular area due to
   a. anterior recession of gingiva
   b. posterior recession of gingiva
   c. paucity of gingivae in second-third molar area
6. Abutment teeth in fixed prosthesis
7. Following gingivoplasty or gingivectomy
8. Tendency toward proliferation of gingival tissue due to mouthbreathing, etc.

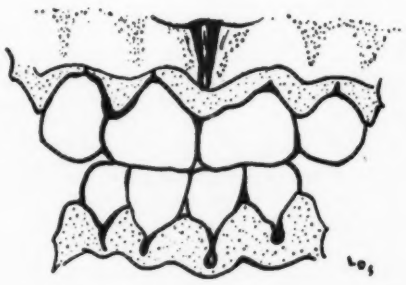

Fig. 6. In the enlargement of gingival tissue due to mouthbreathing, dilantin, etc., the Charters method is most advantageous.

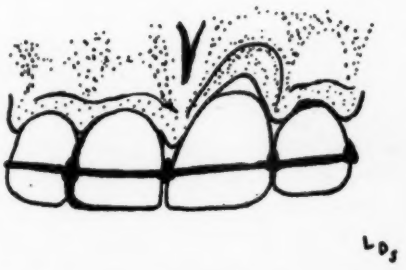

Fig. 8. Temporary splints prevent the bristles from reaching the tooth structure and soft tissues gingivally to the splint. The Charters method enables the bristles to perform satisfactorily and reduce food traps thereby producing better tissue tone.

9. Crater effect of interproximate gingivae following necrotizing ulcerative gingivitis
10. Where wire or acrylic or a combination of both is being employed for temporary splinting.

CONTRA-INDICATIONS

Where there are full interproximate papillae supported by normal interseptal bony crests, it is best to use the modified Stillman method of toothbrush massage.

RESULTS

Specific areas that failed to respond after months of toothbrushing with the modified Stillman method showed marked improvement in tissue tone after two to three weeks of toothbrush massage with the Charters method.

SUMMARY AND CONCLUSIONS

1. Several gingival areas that failed to respond with the modified Stillman method of toothbrush massage, when supplemented by the Charters method, healed most favorably.

2. Anatomic variations that responded favorably with supplementation of the Charters method are noted.

3. There is a need for careful evaluation of the method employed in toothbrush massage for each patient in order to attain maximum benefits in oral health. Charters method of toothbrush massage was employed on the labial only, not as a substitute for, but as a supplement to the Stillman method.

4. The modified Stillman method of toothbrush massage has been found to be satisfactory for the vast majority of our patients at New York University College of Dentistry. However, in this study, emphasis is placed on the fact that there are many variations in architecture that necessitate alterations in toothbrushing procedure.

Grateful acknowledgement is made to Lewis D. Steingesser, N. Y. U. College of Dentistry, 1956, for his art work.

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Mitotic Rate of Gingival Epithelium in Two Age Groups*

by AMARJIT MARWAH, JULIA MEYER, AND J. P. WEINMANN, *Chicago, Illinois*

BULLOUGH[1] observed in 1949 that in middle aged mice the rate of cell division in the epidermis of the ear was about 50% higher than in young adults, even though these had not yet quite reached full size. Thuringer and Cooper[4] found age changes in the same direction in human epidermis but did not report quantitative data. Katzberg[2] found a doubling of mitotic index from the first to eighth decade in human abdominal epidermis.

Mitotic activity in attached gingiva of two age groups (25 to 35 years and 50 to 78 years old males) which neither clinically nor microscopically showed inflammatory changes, was studied. The specimens were excised during the late hours of the morning or early hours of the afternoon and fixed immediately in Zenker's formalin solution. The number of cells in mitosis was counted in one hematoxylin-eosin stained section of each specimen, and the total number of cells estimated from cell counts in sample areas and from measurements of the surface area of the whole section. Thirty specimens of each age group were studied. Four hundred and forty-three mitoses were found in 328,600 cells.

In the group aged 25 to 35 years, mitoses averaged 0.98 cells per 1,000, in the older group 1.56 cells per 1000. This difference was statistically significant. The present study thus shows that an increase in mitotic frequency of the same order as in mice occurs between the ages of 25 to 35 years and 50 to 78 years in the epithelium of the human gingiva.

Contrary to the statement made by Rothman[3] that parakeratosis in the epidermis indicates faster regeneration, no correlation between absence or presence of parakeratosis and mitotic rate was found in the gingiva.

In epithelia containing a granular layer, cell division was only half as frequent as in specimens in which this stage was absent. This difference was statistically highly significant. It was surprising to find the presence of a granular layer associated with a markedly lower mitotic rate, especially in view of the fact that granulation was slight in most specimens and almost negligible in some.

The frequency of cell division in the gingiva was higher than that of the epidermis. An increase in mitotic rate may be noted from the heavily granulated epidermal epithelium with lowest mitotic index, the feebly granulated specimens of gingiva with intermediary mitotic index and agranular gingiva with highest mitotic index.

It is concluded that in epithelia, which maintain their integrity by division of cells at the base and shedding at the surface, this regenerative process is accelerated with increasing age. It may be that emancipation of the cancer cell from the mechanisms which control cell division in normal tissues is facilitated by the weakening of such control in the older individual.

From the Division of Oral Pathology, University of Illinois College of Dentistry.

*A detailed report of the study will appear in the Journal of Investigative Dermatology.

This investigation was supported by Research Grant C2705 from the Division of Research Grants, National Institutes of Health, and by a Tobacco Industry Research Committee Grant.

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The Limitations of a Calculus-Preventing Agent

by B. O. A. THOMAS, D.D.S., PH.D.,* *Palo Alto, California*

THIS brief report is submitted to call to the attention of the dental profession the limitations of a substance which aids in preventing the development of supragingival calculus on the teeth.

For centuries men have been aware of the problems resulting from the accumulation of calculus on the cervical portions of human teeth. Treatment has consisted chiefly of the mechanical removal of such foreign material and the polishing of the involved tooth surfaces. This results temporarily in the return of the gingival tissues from a condition of inflammation to one of health. If scaling and polishing of the teeth is repeated at regular intervals, the periodontal tissues may remain in good condition indefinitely, provided other factors, (local and/or systemic) when present, are recognized and corrected.

Man has long sought for a substance which would eliminate or prevent the formation of dental calculus. Until recently the materials often employed were dilute solutions of mineral acids which dissolved calculus but at the same time decalcified the enamel, cementum, and/or dentin. Much harm was done by such "tartar-preventing" nostrums, which were usually purchased by the lay public for home use without any knowledge of the serious results which followed the application of such solutions. A number of warning articles were published on this subject.[1,2]

Recently an approach to this problem has been reported[3,4,5] which promises more effective and safer results. A material, marketed as EXTAR, by The Parker Company of Philadelphia, Pennsylvania is, if the author's information is correct, an enzyme substance of the mucinase group. The action of this solution is apparently the destruction of the organic matrix of the calculus, thus allowing the mechanical removal of the not yet calcified material from tooth surfaces. It is necessary that the organic material be physically removed by brushing and not merely contacted by the enzyme solution. This requires rather vigorous brushing with a multi-tufted soft nylon toothbrush as specified in directions which accompany the commercial product. Comments have been made by various dentists, questioning the safety of the prolonged use of such substances. While this may not be settled as yet, I am not aware of published evidence to date which indicates harmful effects from this product.

Fig. 1. Lingual surfaces of lower anterior teeth immediately following thorough oral prophylaxis. No calculus present. November 3, 1955.

Following is a brief case report of the author's experience with EXTAR in his own mouth.

For several years it has been my practice to remove the calculus from the lingual surfaces of my lower anterior teeth at intervals of three to four weeks, in order to avoid clinical gingival inflammation.

November 3, 1955: Thorough scaling and polishing of all teeth (see result, Fig. 1). No calculus is present.

January 3, 1956: Two months after the use, morning and evening, of EXTAR solu-

*Formerly Professor and Executive Officer, University of Washington School of Dentistry, Seattle, Washington.

Fig. 2. Same area as Figure 1, two months later. Extar used morning and evening. No apparent calculus. January 3, 1956.

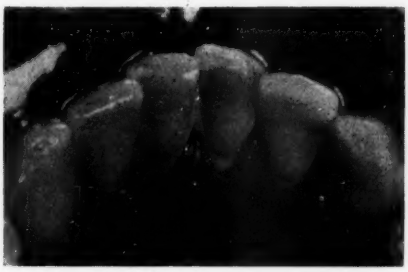

Fig. 4. Same area as Figures 1 and 2, two months after discontinuing the use of Extar. Considerable calculus is evident. March 3, 1956.

tion according to directions (Fig. 2). No apparent calculus is present. However, when the rubber dam was placed, a definite deposition of calculus was found to be present (Fig. 3). The use of EXTAR was discontinued on this date.

March 3, 1956: Two months after discontinuing use of calculus-preventing substance (Fig. 4). It is obvious that much more calculus has developed than had been deposited during the two months when the solution was employed.

COMMENTS

1. The recently developed calculus-preventing substance being marketed as EXTAR seems effective in preventing the deposition of supragingival calculus *in areas which can be mechanically cleansed by the toothbrush.*

2. The safety of this calculus-preventing agent has not been definitely settled, but to date evidence of tooth or soft tissue damage is not available in the literature.

3. Dentists should warn patients for whom such substances seem effective, that calculus formation will still occur in inaccessible areas and that regular scaling and polishing of the teeth is necessary to prevent periodontal disease. Unless this is done, periodontal problems will result from neglect in much the same way as caries problems resulted from neglect following the lay public's misinterpretation of the effectiveness of topical applications of sodium fluoride or the fluoridation of city water supplies.

Fig. 3. Photo taken ten minutes after Figure 2, with rubber dam in place. Facial view to show calculus on proximal surfaces of mandibular incisors. January 3, 1956.

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2. Idem. III. "Bleachodent," "Ekay," "Ex-Tartar," "Snowy-White" and "Tartaroff." Jour. Dent. Res. 10:481, Aug. 1930.

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4. Idem. Clinical Evaluation of a Salivary Calculus Preventive Agent. Oral Surg., Oral Med., Oral Path. 7:607, June 1954.

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Announcements . . . .

ACADEMY OF PERIODONTOLOGY TO MEET SEPTEMBER 27-29, 1956

Dr. Robert G. Kesel, president of the American Academy of Periodontology, writes that: The theme of the program in Atlantic City, September 27-29, is Prevention. Dentistry's primary challenge is the retention of natural teeth—not their replacement, as might be construed from the concern that the profession is demonstrating in dental laboratory operations and the illegal practice of dentistry by laboratory technicians.

"A reduction in tooth loss from periodontal disease could be accomplished in two ways: One, by the wider application of truly preventive measures that would inhibit the initiation of destructive processes; or two, the application of remedial preventive practices applied to check the progress of periodontal disorders. The latter method is extremely important in our human society because so many people do not become patients until disease symptoms are well developed. It is this remedial phase of prevention that our Program Committee has selected as the theme.

"The program is designed to describe technics available for saving periodontally involved teeth based on an understanding of etiology, correct diagnosis, treatment planning and tissue response. An innovation for our meeting will be the dicussion of treatment planning for a problem case. The history, oral examination, photographs of the x-ray films and casts of the dentition will be made available to each member and guest as they register for the meeting so that they may familiarize themselves with the problems involved. Four of our members will discuss various aspects of the case on Saturday morning and will outline their individual plan of treatment. Following their appraisal, questions and alternative suggestions for treatment will be sought from the assembly. This session on treatment planning should be profitable and stimulating.

"The officers and the committees who have worked diligently to make the 42nd Anniversary Meeting attractive and educational invite you to attend. There will be important decisions to make at the business meeting, including consideration of an attendance requirement for the annual meeting. (You will find the preliminary program printed in the July, 1956, issue of *The Journal of Periodontology*). At the same time, Atlantic City is a fine place to relax. We hope to see you there."

TEMPLE UNIVERSITY

Course 1—Periodontics—Dr. Jacoby Rothner and associates. November 5 to November 17 inclusive.

Course 3—Occlusion and What To Do About It—Dr. Bernard Jankelson. December 3 to December 6 inclusive.

Course 6—Periodontal Prosthesis—Dr. Morton Amsterdam and Dr. Walter Cohen. February 11 to February 16 inclusive.

Course 7—Orthodontics in Periodontal Therapy—Dr. Jack Alloy and Dr. Maxwell Fogelman. April 11 to April 13 inclusive.

For additional information and application, write to Dr. Louis Herman, Postgraduate Division, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

UNIVERSITY OF SOUTHERN CALIFORNIA

The University of Southern California, School of Dentistry, announces the following postgraduate course to be presented December 10, 11, 12, 14 and 15, 1956:

Histopathology of the Periodontium. Instructor: Balint Orban, M.D., D.D.S., assisted by Nathan Friedman, B.S.D., D.D.S., Professor of Periodontology. Tuition fee: $150.00.

For details write to the Division of Postgraduate Instruction, University Park, Los Angeles 7, California.

NORTHWESTERN UNIVERSITY

Periodontics for the General Practitioner—I

October 17, 24, 31, November 14, 21, 28, December 12, 19, 1956, January 16, 30, February 20, March 6, 20, April 3, 17, May 1, 15, 29, June 12, 1957.

Faculty: G. R. Lundquist, D.D.S., M.S., Professor of Oral Pathology and Chairman of the Department of Clinical Oral Pathology. Donovan W. Brown, D.D.S., M.S., Professor of Oral Pathology. Arthur Elfenbaum, B.A., D.D.S., Professor of Diagnosis. Harry Sicher, M.D., Guest Lecturer. Professor of Anatomy, Loyola University School of Dentistry. Max Gratzinger, M.D., D.D.S., M.S., Associate Professor of Oral Pathology (Course Leader). Irvin T. Harris, D.D.S., M.S.D., Associate Professor of Oral Pathology.

This extensive course is designed to develop additional knowledge and skill to enable the practitioner to cope successfully with the periodontal problems of his patients. The necessary background for diagnosis, prognosis, and treatment planning will be provided, as well as the opportunity to acquire skill in all phases of treatment, including surgical and orthodontic procedures with emphasis on removable plate appliances. Patients can be provided by the participants for consultation and treatment or clinical cases will be made available. Tuition: $500.00.

Periodontics for the General Practitioner—II

September 19, October 10, November 7, December 5, 1956, January 9, February 13, March 13, April 10, May 8, June 5, 1957.

Faculty: G. R. Lundquist, D.D.S., M.S., Professor of Oral Pathology and Chairman of the Department of Clinical Oral Pathology. Donovan W. Brown, D.D.S., M.S., Professor of Oral Pathology. Max Gratzinger, M.D., D.D.S., M.S., Associate Professor of Oral Pathology (Course Leader). Irvin T. Harris, D.D.S., M.S.D., Associate Professor of Oral Pathology.

Periodontics for the General Practitioner—I is prerequisite to this advanced course in periodontics. The class will meet once a month for further study and discussion. The current literature will be critically evaluated. As in the previous course, the treatment of patients principally furnished by the participants will be considered further. Tuition: $200.00.

Clinical Management of Gingivitis and Periodontitis

January 28, 29, 30, 31, February 1, 1957.

Faculty: G. R. Lundquist, D.D.S., M.S., Professor of Oral Pathology and Chairman of the Department of Clinical Oral Pathology. Donovan W. Brown, D.D.S., M.S., Pro-

fessor of Oral Pathology. Joseph C. Calandra, Ph.D., M.D., Professor of Pathology and Bacteriology. Stanley C. Harris, Ph.D., Professor of Physiology and Pharmacology. J. Robert Schumaker, D.D.S., M.S.D., Professor of Histology. Richard W. Tiecke, D.D.S., M.S., Associate Professor of Pathology.

This course emphasizes consideration of the most common forms of periodontal disease. A short review will be included of the pertinent basic sciences as they are correlated with the etiology, diagnosis, treatment, and prognosis of the different diseases involved. Local and systemic factors will be studied comprehensively.

All communications regarding these courses should be addressed to Director of Dental Postgraduate Study, Northwestern University Dental School, 311 East Chicago Avenue, Chicago 11, Illinois.

TUFTS UNIVERSITY

Tufts University School of Dental Medicine announces the following Postgraduate Refresher Course, in addition to those mentioned on pages 235-236 of the July issue of this Journal:

DPG 605—Occlusal Equilibration. Monday, Tuesday, Wednesday, Feb. 11, 12, 13, 1957. Tuition: $100.00—Class limited to twelve. Dr. Edward T. Fischer with Dr. Sidney Sorrin, New York University.

This is a practical course devoted to the detection and correction of trauma from occlusion. It is a course for the general practitioner, the periodontist, the orthodontist and the prosthodontist. Technics for detecting occlusal disharmonies will be analyzed and demonstrated on patients. The technic for equilibrating the occlusion will be described in lecture, demonstrated on models and performed on patients.

For further information and application write to: Director of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Massachusetts.

UNIVERSITY OF PENNSYLVANIA

The following postgraduate courses may be of interest to the readers of this Journal.

Dr. Charles L. Pincus from Beverly Hills, California—Esthetic Variations in Jacket Crown and Bridge Restorations involving Periodontal Lesions, 8th, 9th, 10th October, 1956. This course will cover the technical procedures emphasized in multiple jacket crown and bridge restorations with special emphasis on esthetics.

Dr. Henry M. Goldman and Dr. D. Walter Cohen—Periodontal Therapy—10th to 14 December, 1956. The objective of this course is to acquaint the practitioner with the importance of periodontics in the general practice of dentistry.

Dr. Manuel M. Album—Dentistry for Handicapped Children—12th to 16th December, 1956. The objective of this course is to acquaint the practitioner with the importance of periodontics in the general practice of dentistry.

For further information concerning these courses please communicate with Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4, Pennsylvania.

OHIO STATE UNIVERSITY

The Ohio State University, College of Dentistry, will present the following postgraduate courses during 1956-1957:

Anatomy of the Head and Neck—Linden F. Edwards. Sept. 24-28 and March 18-22.
Endodontics—J. Henry Kaiser. Jan. 21-25.
Oral Diagnosis—Hamilton B. G. Robinson. Jan. 28-Feb. 1.
Partial Denture Prosthodontics—Victor L. Steffel. Feb. 18-22.
Review of Clinical Dentistry—Dental Faculty. May 13-17.

Each of these courses is presented for five consecutive days (Monday through Friday) and the fee is $50 per course with the exception of Oral Surgery and Endodontics for which a fee of $100 is charged. Further information may be secured from the College of Dentistry, Ohio State University, Columbus 10, Ohio.

THE ROWE SMITH MEMORIAL FOUNDATION
announces
THE THIRD ANNUAL SEMINAR

Subject: The Problems of Pain in Dental Practice.

Moderator: E. Cheraskin, M.D., D.M.D., Chairman of the Department of Oral Medicine, University of Alabama School of Dentistry. Co-author of *The Physiological Foundation of Dental Practice, The Physiology of Man, and Dynamics of Oral Diagnosis.*

The Essayists are: Ruth Moulton, M.D., Assistant Clinical Professor of Psychiatry, Columbia University, and practicing psychoanalyst. Stewart Wolf, M.D., Professor and head of the Department of Medicine, University of Oklahoma Medical School. Consulting Professor of Neurology and Psychiatry. Head of Psychosomatic and Neuromuscular Section at the Oklahoma Research Foundation.

Topics: The Anatomy and Physiology of Pain, The Psychology of Pain, Trigeminal Neuralgias, Primary Atypical Facial Neuralgia: A Conversion Hysteria, Pain of Maxillary Sinus Origin, The Role of the Oral Cavity in Psychosexual Development, Headaches of Dental Origin, Temporomandibular Joint Pain, Cardiac Pain, Local Anesthetic Agents, Analgesic Agents, The Psychology of Anesthesia.

Time: December 2, 3, 4, 5.

Place: Fly-n-Fish Lodge, Caddo Lake, Karnack, Texas.

Fee: $175.00 (This includes tuition and room and board.)

Attendance will be limited.

Further information available from Rowe Smith Memorial Foundation, 501 State National Bank Building, Texarkana, Arkansas.

Sorry, Please!

In the announcement of the program for the Saturday afternoon clinics (to be presented as a feature of the meeting of the Academy of Periodontology Sept. 27-29) in the July issue of this Journal (page 233) the first presentation was erroneously listed as by Goldberg, Goldberg, and Bibby. It should have been H. J. V. Goldberg, L. Goldenberg, and B. G. Bibby. Sorry!

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Eleventh Mid-Annual Meeting and Luncheon at the Hotel New Yorker in New York City on Sunday, December 2nd, 1956. There will be a business meeting for members at 10:30 a.m., followed by a luncheon for Academy members and friends at 12:30 p.m.

The afternoon session will be devoted to a symposium on *Recent Concepts on the Temporomandibular Joint in Relation to Fixed and Removable Dentures and Roentgenographic Interpretation.*

All members and interested dentists and physicians are cordially invited to attend. Address: Dr. A. Reiner, Chairman, 114-01—201st Street, St. Albans 12, N. Y., or Dr. Andrew Cannistraci, Treasurer, 2152 Muliner Avenue, Bronx, N. Y.

AMERICAN BOARD OF ORAL MEDICINE ELECTS OFFICERS; ADOPTS CERTIFICATION REQUIREMENTS

Election of officers and adoption of certification requirements featured the meeting of the American Board of Oral Medicine held concurrently with the tenth annual session of the American Academy of Dental Medicine at the Sheraton-Cadillac Hotel, Detroit, Mich., June 22-24.

The Board is composed of the following: Dr. Hermann Becks (M.D., D.D.S.), Professor of Oral Medicine, University of California, San Francisco; Dr. Lester W. Burket (M.D., D.D.S.), Dean, School of Dentistry, University of Pennsylvania, Philadelphia; Dr. Harold R. Gelhaar (D.D.S.), Upper Montclair, N. J., a past president of the American Academy of Dental Medicine; Dr. Irving Glickman (D.M.D.), Associate Dean, Tufts College Dental School, Boston, Mass.; Dr. Samuel Charles Miller (D.D.S.), Professor of Periodontia and Oral Medicine, New York University College of Dentistry, New York, N. Y.; Dr. Joseph F. Volker (D.M.D.), Dean, School of Dentistry, University of Alabama, Birmingham.

The following officers were elected: Dr. Miller, Chairman, Dr. Burket, Secretary, and Dr. Gelhaar, Treasurer.

At its meeting the Board adopted By-laws and the following requirements for certification: A minimum of two years of advanced study at a recognized educational institution; three years of practical experience, and successful completion of examinations of the Board, which will include written, oral and practical examinations, as well as five case reports.

Those interested in further information regarding certification in oral medicine are invited to apply to the Secretary.

Letter to the Editor • • • •

A current article published in the Journal (Shapiro, Max: Orthodontic Procedures in the Care of the Periodontal Patient, J. Periodont., 27:7-15, Jan. 1956) contained some clinical observations and conclusions which, I feel, bear reexamination.

In the case described as "number 8," the labial arch wire on the upper anterior segment of teeth was so contoured as to interfere with the continuous eruption of the upper left central tooth. The arch wire being obviously placed so that it crossed incisally to the most labial contour of the tooth. Figure I. Further description in the text indicates that as soon as the wire was repositioned, the upper left central tooth was permitted to "catch up" with its contiguous teeth. Figure IB

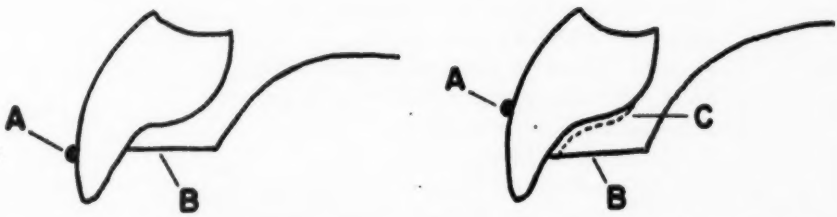

Fig. I. A. Labial arch wire positioned incisally to the most Labial contour of the tooth. B. Table of Bite Plane.
Fig. IB. A. Labial arch wire positioned normally. B. Table of Bite Plane. C. Tooth surface of appliance contoured to permit continuous eruption.

The text further concludes that case No. 8 illustrates "a technique by which teeth may be intruded where necessary." I believe that the clinical behavior of the case created the optical illusion of intrusion of the upper left central tooth; instead interference with the biologic mechanism of continuous eruption of teeth was responsible for this clinical misinterpretation.

Space does not permit a more detailed discussion of the use of my recommendations for the use of the bite plane as an adjunct in the treatment of periodontal disease. Anyone interested may find a detailed report in my handbook, *Clinical Periodontology: Dynamics and Treatment*, Park Press, 1953, New York City and in a motion picture released in 1955 which was shown at the last National Dental and Chicago Midwinter Meetings.

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PERIODONTIST'S SOLILOQUY

A PARODY*

To treat or not to treat: that is the question:
Whether 'tis nobler for the patient to suffer
The pain and anguish of lengthy treatment
Or to accept the forceps on some weakened teeth
And by extracting end the problem. To scale, to polish
No more; and by extraction end
The trouble, and the thousand natural shocks
That oral flesh is heir to, 'tis a consummation
Often to be wish'd. To splint, to cut,
To pack; perchance to fail: aye, there's the rub;
For in that failure to succeed what criticisms may come,
When patients return with still
Mobile teeth and bleeding gums . . . . .
Then we would bear the whips and scorns of time.
"The dentist's wrong," the patient then would say
And for the pain and anguish
The patient still must pay and pay.
The patient notes the bad result
And states that he himself had wanted dentures.
Patients have no knowledge of what follows loss of teeth.
The unknown edentulous state from whose bourn
No patient can return, even with the aid of Dr. Sicher.
So we treat periodontal disease
And makes us rather bear those ills we have
Than fly to others in the denture field
We do not choose to join the prosthodontists clans.
Thus conscience (and Orban) does make periodontists of us all.

M.K.H.

*With apologies to several, including Shakespeare, Hamlet and the readers, if any. Read at the Colorado Foundation's Tenth Annual Seminar, July 5, 1956.

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